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UNIVERSITY OF ALBERTA

Biofeedback Treatment of Temporomandibular Joint Dysfunction

BY

Roger O. Gervais ©

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE
OF Doctor of Philosophy

Department of Educational Psychology

EDMONTON, ALBERTA
Spring 1991

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Dedication

This work is dedicated to my parents whose love, support, and encouragement have been constant throughout all of my academic career.

Abstract

The present study contrasted the effectiveness of two biofeedback training schedules, as compared to progressive relaxation, in treating 100 female TMJ pain-dysfunction patients. The patients were assigned to one of three treatment groups or a no-treatment control condition: weekly biofeedback (N=26), daily biofeedback (N=25), progressive relaxation (N=24), and control (N=25). Four measures of treatment outcome were obtained: (a) pre and posttreatment EMG activity from the temporalis and masseter muscles, (b) pre and posttreatment clinically assessed TMJ and muscle palpation pain and noise, (c) daily pain symptom chart, and (d) subjective percentage improvement following treatment.

MANOVAs conducted on the pre and posttreatment EMG and clinical measures determined that all groups demonstrated significantly reduced EMG activity at the posttreatment assessment. There was no statistically significant difference in mean temporalis and masseter EMG between the groups. All patients showed significant reductions in clinical palpation pain and joint noise at posttreatment, but no significant between-groups differences.

Despite the significant reduction in clinically assessed palpation pain, there was no significant change over time in self-reported pain symptom intensity as registered on the daily symptom chart. The weekly biofeedback group reported a significantly greater 45.38% improvement in symptoms following treatment than did the daily biofeedback group (26.20%) or the relaxation group (27.38%)($p<.05$).

It was concluded that none of the treatment modalities was significantly superior to the no-treatment control condition in reducing the clinical signs and symptoms of TMJ pain and dysfunction. The weekly biofeedback schedule did, however, produce a statistically greater degree of subjective improvement than did the daily schedule, the relaxation program or the control condition. Limitations of the study, implications for the application of biofeedback in TMJ disorders, and directions for future research are discussed.

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I. INTRODUCTION

Electromyographic (EMG) biofeedback training has gained widespread acceptance in the treatment or management of a wide range of disorders. The recognition that formerly hidden or inaccessible physiologic processes can potentially be brought under conscious voluntary control has revolutionized the conventional models of health care, and has expanded the role of the psychological profession in promoting wellness. As in other areas of health care, the multidisciplinary exchange between psychology and dentistry has grown steadily in the last two decades. A primary focus of this professional collaboration has been in the treatment of temporomandibular joint dysfunction and pain (TMJ), often using biofeedback or some other form of relaxation training (Moss & Garrett, 1984).

Temporomandibular pain and dysfunction disorders are increasingly being viewed as a category of disturbances, of multifactorial etiology, affecting the masticatory musculature and temporomandibular joints. The literature generally agrees that physical factors interact with psychological characteristics and psychophysiologic predisposition leading to the emergence of clinical signs and symptoms. The psychophysiologic theory of TMJ pain dysfunction as developed by Schwartz (1955, 1956, 1958; Schwartz & Cobin, 1957); and expanded by Laskin (1969, 1980), and Laskin and Block (1986), with the definition of the myofascial pain-dysfunction (MPD) syndrome, provides one of the major theoretical frameworks for understanding the etiology, symptomatology, and treatment of temporomandibular disorders. In this view, the patient's musculoskeletal response to stress is a central precipitating factor in the emergence of clinical signs and symptoms (Greene, 1980). Accordingly, neuromuscular hyperactivity and pain in the masticatory muscles is invariably observed.

With the collaboration between dentistry and psychology has come interest in using behavioural medicine techniques as an adjunct to conventional dental treatment for temporomandibular disorders. Biofeedback training aimed at promoting self-awareness and self-regulation of masticatory muscle activity in the TMJ patient is the most frequently used psychological intervention. Since Solberg and Rugh (1972) published one of the first accounts of biofeedback training with a groups of TMJ patients, there has been continuing interest in this application as evidenced by a growing body of research and clinical literature.

The majority of published reports endorse biofeedback training as effective in regulating EMG activity in the target muscle. This is generally accompanied by a decrease in subjective, self-report symptoms. Conversely, there have been no conclusive findings with respect to changes in clinical signs of mandibular dysfunction following biofeedback training (Moss & Garrett, 1984). Consequently, no specific treatment effect has been identified to account for the success of biofeedback training in temporomandibular dysfunction. Attempts have been made to isolate specific treatment effects including: increase in perceived control (Hijzen, Slangen, & van Houweligen, 1986; Stenn, Mothersill, & Brooke, 1979); muscular relaxation and self-regulation (Dalen, Ellersten, Espelid, & Gronningsaeter, 1986: Carlsson & Gale, 1976); and various non-specific factors such as the interaction between patient motivation, psychophysiological idiosyncracies, and the treatment setting (Dahlström, Carlsson, Gale, & Jansson, 1984).

Moss and Garrett (1984), in a comprehensive review of the TMJ dysfunction syndrome, make a number of recommendations for further research into the use of biofeedback in this application. Among others, they identify the following research priorities:

1. There is a need for more detailed and specific clinical examination of patients reporting the same

symptoms in order to more precisely discriminate between groups of subjects.

2. A body of normative data on EMG levels in the masticatory muscles is needed for assessment and treatment research.
3. Well-controlled treatment outcome studies are needed to evaluate the efficacy of the treatment procedures for specific patient symptoms.

In addition to the above recommendations, there is also a need for more investigation of the specific treatment effects underlying the success of biofeedback training in mandibular disorders. This also includes research into the relationship between EMG activity in the masticatory musculature, reported pain, and the clinical signs and symptoms of temporomandibular disorders.

The present study builds upon previous research in a number of ways. With respect to recommendations 1 and 2 from Moss and Garrett (1984), Gervais (1984) reported EMG levels from the temporalis and masseter muscles for two groups of subjects, asymptomatic and TMJ, at baseline, and throughout a series of mental and physical stressors. There were no significant differences between the two groups with respect to EMG activity, however, both groups varied significantly from baseline during the stress conditions. A self-report symptom questionnaire discriminated between both groups, and the subjects in the TMJ group were given a thorough clinical examination for the purpose of recording the signs and symptoms of mandibular dysfunction. For both groups there were significant correlations between EMG activity and the number of positive responses on the self-report questionnaire. In the TMJ group, there were significant, or near significant, correlations between EMG activity and clinical palpation pain scores for seven of nine experimental conditions.

In a subsequent study (Gervais, Fitzsimmons, & Thomas, 1989), the EMG data from the asymptomatic and TMJ (renamed subclinical) groups described above were contrasted with those from a sample of clinical TMJ patients referred for bioreedback training. Whereas there was no significant difference in baseline EMG activity between the first two groups, the patient group demonstrated significantly higher muscle activity (p<.001) than either the asymptomatic or subclinical groups. These findings were presented as preliminary EMG norms to discriminate between TMJ patients with a significant neuromuscular component in their condition, and other categories of patients or non-patients. It was also proposed that the significantly elevated EMG activity noted in the patient group was a major contributing factor in the emergence of the clinical symptoms and the transition from subclinical to patient status. The need for additional research into the relationship between EMG activity and the clinical signs and symptoms of mandibular dysfunction was emphasized by the findings of this study.

A. Overview

The present study was conceived primarily as an evaluation of the effectiveness of variations on biofeedback training schedules in the treatment of temporomandibular disorder patients. A secondary emphasis was placed on clarifying the relationship between EMG activity in the masticatory muscles and the clinical signs and symptoms of dysfunction and pain. It was hoped thereby to add to the knowledge of specific treatment effects responsible for the success of this application of biofeedback training.

As described above, there is substantial evidence in the literature that biofeedback training is effective in reducing the symptoms of temporomandibular dysfunction. What is lacking, however, are studies contrasting the effectiveness of different biofeedback training protocols. The present study compared the subjectively reported and clinically assessed outcome obtained following two biofeedback protocols, progressive relaxation, and a no-treatment control condition.

The two biofeedback protocols consisted of five identical training sessions, each one hour in length. The only difference between the protocols was the interval between biofeedback sessions. The weekly biofeedback group attended one training session per week over a period of five weeks. The daily biofeedback group received one training session daily for five consecutive days. Both biofeedback groups practiced progressive relaxation at home concurrently during the course of the biofeedback training. The relaxation group practiced progressive relaxation at home for five weeks, but received no biofeedback. The control group received neither biofeedback nor relaxation training during the five week control period.

All participants received pre and posttreatment clinical examinations during which the nature and severity of mandibular dysfunction signs and symptoms were recorded. The research design incorporated a pre and posttreatment four (conditions) by four (groups) repeated measures design for gathering the EMG data. The repeated measures consisted of independent EMG recordings from each of the temporalis and masseter muscles at baseline, and during three mental and physical stressors. Pretreatment personality characteristics were also assessed using the Minnesota Multiphasic Personality Inventory (MMPI) and the Myers-Briggs Type Indicator (MBTI). Patients were sequentially assigned to the treatment groups as the referrals to the study were received.

The statistical analysis was undertaken in three stages. The first analysis used multivariate analysis of variance (MANOVA) procedures to test for significant within and between groups differences in the pre and posttreatment EMG repeated measures.

The second analysis used MANOVA to contrast the pre and posttreatment EMG repeated measures for significant Group x Time effects. Multivariate procedures were also used to contrast the pre and posttreatment pain and dysfunction scores obtained in the clinical examinations, as well as the subjective daily pain rating for each week of the study.

The third stage of the statistical analysis involved calculating Pearson product correlations between; a. pre and posttreatment EMG levels and the pre and posttreatment clinical pain and dysfunction scores, b. pre and posttreatment EMG activity and subjective daily pain rating, c. pre and posttreatment EMG and MMPI scale T scores, d. MMPI scale T scores and the pre and posttreatment clinical pain and dysfunction scores and, e. subjective percentage improvement and pre and posttreatment EMG, clinical pain and dysfunction, and MMPI scale T scores.

B. Purpose

The principle hypothesis being tested in the present study is that simultaneous four site EMG biofeedback training from the temporalis and masseter muscles will be more effective in reducing the signs and symptoms of temporomandibular dysfunction in a sample of clinical patients than either home-based relaxation training or a no-treatment control condition. The secondary purpose of this study is to evaluate whether the five hour biofeedback program is more effective when provided once per week for five weeks, or daily for five consecutive days.

It is expected that the biofeedback groups will demonstrate significantly reduced posttreatment EMG levels during baseline and experimental stress as compared to pretreatment EMG levels. Significant differences in pre and posttreatment clinical pain and dysfunction scores, and subjective daily pain rating are expected in the three treatment groups, but not in the control condition. The degree of reduction in clinical pain and dysfunction, and subjective daily pain rating is expected to be greater in the biofeedback groups. Accordingly, posttreatment self-report percentage improvement is expected to be greater in the biofeedback conditions than in the relaxation condition.

Significant correlations are expected between the number of positive self-report symptoms and clinical signs of mandibular pain-dysfunction and the magnitude of EMG activity in the temporalis and masseter muscles. It is also anticipated that successful treatment outcome, as measured by a reduction in clinical pain and dysfunction and a positive self-report percent improvement, will correlate significantly with learned ability to reduce temporalis and masseter EMG activity.

It is anticipated that the present study will corroborate the conclusions of the research literature that EMG biofeedback training is an effective adjunct therapy in the management of temporomandibular pain-dysfunction disorders. The conclusions of this study will explore the nature of specific treatment effects and provide direction for further developments in the use of biofeedback therapy in temporomandibular disorders.

II. REVIEW OF THE LITERATURE

A. TMJ/MPD

History of the TMJ/MPD Syndrome

In 1934, J.B. Costen, an otolaryngologist, published an article which presented the cases of 11 patients suffering from a variety of "ear and sinus" symptoms which he claimed were due to "disturbed function of the temporomandibular joint" (Costen, 1934). The symptoms he observed consisted of impaired hearing, a stuffy or stopped sensation in the ears, tinnitus, pain within and about the ears, dizziness, severe headache, and burning sensations in the throat, tongue, and nose. Costen attributed these symptoms to some disorder or irregularity in the anatomic function of the temporomandibular joints, their connective tissues and muscles due to a collapse in vertical dimension resulting from loss of posterior tooth support. In treating these patients, Costen claimed that these symptoms were relieved in nine of the eleven cases by prosthetic dentistry to open the bite.

Costen's work provided a theoretical link between these previously unrelated symptoms which had long puzzled researchers, and the direct mechanical function of the temporomandibular apparatus. In the decade and a half that followed Costen's article, however, a number of authors criticized his theory on the basis of clinical treatment outcome and anatomical research (Guralnick, Kaban, & Merrill, 1978). During this period Costen's original list of symptoms was modified as some were dropped as being unrelated to TMJ function, while others were added.

In the 1950s Laszlo Schwartz and his colleagues redefined "Costen's syndrome", as it was commonly known, and termed it the "pain-dysfunction syndrome" (Schwartz, 1955, 1956, 1958, 1959). Schwartz considered the symptoms relating to the TM-joint analogous to those surrounding other joints in the body. Pain in the temporomandibular area, as well as the other related symptoms, was attributed to incoordination of the muscles of mastication. Muscular dysfunction was followed by muscle spasm which in turn led to a "persistent pain-spasm cycle" (Guralnick et al., 1978). Schwartz also considered the patient's occlusion, dental history, and psychological predisposition to be significant contributing factors in the onset and development of the pain-dysfunction syndrome. At this point, a theoretical understanding of TMJ disorders which encompassed both anatomy and physiology as well as psychology, began to replace "Costen's Syndrome" with its purely mechanical perspective on facial pain.

A further development in the theory of temporomandibular joint pain came with the work of Laskin, who in the late 1960s proposed the myofascial pain-dysfunction (MPD) theory. This theory extended the Schwartz's concept by viewing masticatory muscle spasm initiated by muscular over-extension, over-contracture, or muscle fatigue, as the primary factor in the onset of pain (Laskin, 1969). The muscle spasm could be precipitated by a number of factors including malocclusion and muscle fatigue produced by chronic oral habits such as bruxing or clenching the teeth.

In the twenty years since Laskin (1969) revised the concept of the TMJ pain dysfunction syndrome, there has been considerable multidisciplinary interest in the condition, its definition, clinical diagnosis, and treatment. Whereas Laskin's concept of MPD continues to emphasize the primarily psychophysiologic nature of the disorder (Laskin, 1986), Ash (1986), uses the term "TMJ and muscle dysfunction syndrome" generically to denote a collection of signs and symptoms associated with structural and/or functional disorders of the temporomandibular joints and associated musculature. There is no implication that the disturbance must be primarily psychophysiological, nor entirely organic (Ash, 1986).

A summary review of the recent clinical and research literature finds temporomandibular disorders referred to by a variety of names: TMJPDS (temporo-mandibular pain and dysfunction syndrome, Salter et al., 1983); CMD (craniomandibular dysfunction, Wabeke et al., 1989); TMJS (temporomandibular joint dysfunction syndrome, Schumann et al. 1988); TMJDS (temporomandibular joint disturbance syndrome, Chong-Shang & Hui-Yun, 1989); and TMD (temporomandibular disorders, Pullinger & Monteiro, 1988). Each name attempts to more precisely define the condition according to the particular research or treatment orientation of the investigator or clinician. The term 'craniomandibular syndrome', or some variation, is receiving increasing usage (Bell, 1986).

Predictably, the multiplicity of labels in the temporomandibular joint pain dysfunction nomenclature, and the varying and often competing theoretical positions on the etiology and treatment of the disorder, have given rise to criticism of the concept of the temporomandibular joint pain dysfunction syndrome. In general, the trend is to move away from the syndrome concept of temporomandibular joint disorders in favor of a multifactorial view of the variety of conditions which can afflict the joint and associated musculature (Bell, 1986; Reynolds, 1988).

Anatomy

The structural and functional anatomy of the temporomandibular joint has been the subject of investigation and debate for many years. Rees (1954), in a meticulous and enduring anatomical study of the temporomandibular joint, provided perspectives on the functional dynamics of the joint, the influence of which continue to be seen in contemporary discussions of anatomy (Yung, Carpentier, Marguelles-Bonnet, & Meunissier, 1990).

The temporomandibular joint is a complex synovial joint providing the articulation between the mandible and the cranium. The condyle of the mandible rests within the glenoid or articular fossa and is capable of simultaneous rotatory and sliding movements within and along the fossa. The articulating surfaces are separated by the articular disc which separates the joint space into upper and lower compartments. The rotatory, hinge-like movement of the mandible occurs as a function of the lower joint compartment, while the linear sliding movement is produced as the condyle and articular disc glide forward along the articular eminence which forms the anterior boundary of the articular fossa. Most movements of the jaw involve translatory movement which is a combination of rotatory movement in the lower joint compartment and sliding in the upper joint (Bell, 1986). This characteristic feature of the temporomandibular joint has led it to be classified as a hinge joint with a moveable socket (Hylander, 1980).

The temporomandibular joint is enclosed by a fibrous capsule which is lined by synovial membranes. It is attached to the temporal bone along the border of the articular fossa and eminence anteriorly, medially, posteriorly, and laterally. It extends from the temporal bone to the mandibular neck and is strongly reinforced laterally by the temporomandibular ligament. The temporomandibular ligament serves to limit the movements of the mandible, and, in particular, to prevent displacement of the condyle away from the articulating surfaces and against the posterior portion of the joint which could damage the posterior disc attachments and associated tissues (Dolwick & Sanders, 1985). In contrast to the thick, tough temporomandibular ligament reinforcing the lateral wall of the articular capsule, the anterior, medial, and posterior walls are relatively thin and loose. The articular capsule also fulfills a vital role in assuring the lubrication and nourishment of the articular surfaces by containing and distributing the synovial fluid throughout the two joint cavities (Bell, 1986).

The articular disc separating the joint compartments is composed of dense, avascular fibrous tissue. Along its anterior border it fuses with the joint capsule, and posteriorly, it merges into the retrodiscal pad, a thick layer of highly vascularized and innervated connective tissue. The retrodiscal pad attaches to the posterior wall of the capsule and normally lies in loose folds when the jaw is at rest with the condyles centered in the articular fossa. During translatory movements of the condyle/disc complex the retrodiscal tissues are stretched and provide posterior traction to the articular disc which serves to maintain the correct relationship between the disc and the articulating surfaces. Along its medial and lateral edges, the articular disc attaches firmly and independently to the condyle (Dolwick & Sanders, 1985).

The temporomandibular joint and capsule are innervated by the articular branches of the auriculotemporal, masseteric, and posterior deep temporal nerves (Kawamura, 1980). These nerves extend throughout the joint capsule and terminate at the periphery of the articular disc, leaving the central portion of the disc without innervation. The auriculotemporal nerve, deriving from the mandibular division of the trigeminal nerve is the primary sensory nerve in the temporomandibular joint, and provides afferent signaling for proprioception and nociception in the highly sensitive posterior joint space and capsule including the retrodiscal pad. The anterior joint and capsule are innervated by the masseteric and temporal nerves (Bell, 1986). Autonomic fibres from the auriculotemporal nerve also enter the capsule medially and supply the fine blood vessels in the joint (Kawamura, 1980).

The masseter, temporalis, medial pterygoid, and lateral pterygoid are the four muscles of mastication. The first three are elevator muscles and function primarily to close the jaws. Protrusion, lateral movements, and jaw opening are related to lateral pterygoid activity.

The lateral pterygoid consists of two functionally distinct and independent parts: the superior and inferior heads. The inferior head attaches to the mandibular neck and is active in protraction, opening, and lateral excursion. The superior head is attached to the disc and contracts during elevation of the mandible and provides constant anterior traction on the articular disc during the translatory cycle to counter balance the posterior traction supplied by the retrodiscal tissues. In this manner, the stability of the joint is maintained throughout the range of normal mandibular movement (Bell, 1986). The lateral pterygoid is innervated by branches of the masseteric or buccal nerves (Hylander, 1980).

The masseter and temporalis, along with the medial pterygoid, are the masticatory muscles responsible for elevation of the mandible. The masseter is a strong rectangularly shaped muscle extending from the zygomatic arch to the outer surface of the mandibular ramus. Its primary function is to elevate the mandible and deliver maximum force during the power strokes of the masticatory cycle. The masseter is innervated by the masseteric nerve.

The medial pterygoid is a functional counterpart of the masseter muscle and is located on the medial side of the mandibular ramus. At the point of insertion, its fibres often meet fibres of the masseter beneath the mandibular angle. It is innervated by the medial pterygoid nerve which branches off the mandibular division of the trigeminal nerve (Hylander, 1980).

The temporalis is a large fan-shaped muscle whose fibres originate along the lateral surface of the skull and converge to attach into the coronoid process and along the anterior border of the mandibular ramus. Due to the fan-like shape of the muscle, the fibres of the temporalis exert their activity in different directions. The posterior fibres provide a vertical force upon the mandible, whereas the middle fibres exert a retracting force. The anterior fibres of the temporalis may also function in combined protrusive and closing movements of the jaw. The temporalis is innervated by the temporal branches of the mandibular division of the trigeminal nerve (Hylander, 1980).

Symptomatology

Only a few of the symptoms composing Costen's original syndrome have been retained by contemporary TMJ pain-dysfunction theory. Schwartz, in a well-known study of 491 TMJ patients (Schwartz, 1959), found that the three most prevalent symptoms were: pain (75%), clicking (9%), and limitation of mandibular movement (7%). Additionally, pain occurred in conjunction with other symptoms such as clicking and limitation in 62% of the sample. The quality of the pain was described as "a constant unilateral jaw ache, earache, or headache, usually aggravated by mandibular movement" (Schwartz & Chayes, 1966).

A few years later, Laskin clarified what were proposed to be the essential symptoms in the myofascial pain-dysfunction syndrome. As did Schwartz, Laskin emphasized the presence of unilateral pain, described as a constant, dull ache in or about the ear, which can extend generally to the head and neck. The pain is often worse in the morning although it can also augment during the course of the day (Laskin, 1969). Clicking and limitation of opening were also common complaints. To this symptom triad Laskin also added muscle tenderness, a condition of which most patients were not aware.

At present there appears to be general agreement upon the four principle symptoms relating to the myofascial pain-dysfunction syndrome: 1. Dull ache or pain, anterior to the ear and sometimes involving the face, head and neck. The pain may be predominantly unilateral, but not necessarily so. 2. Clicking of one or both joints, 3. Limitation of mandibular movement, 4. Muscle tenderness (Brooke, Stenn, & Mothersill, 1977). Other researchers have found additional symptoms to be associated with the syndrome. These include chronic minor illness such as migraine, back, neck or shoulder pain, skin disorders, hay fever, and asthma (Berry, 1969); recurrent headaches (Magnusson & Carlsson, 1978); stuffiness in the ear, hearing loss, dizziness and disequilibrium (Koskinen, Paavolainen, Raivio, M., & Roschier, J. 1980; Weinberg, 1980); tinnitus, blurred or double vision, and change of voice (Reade, 1984). However, for the most part, the symptoms most commonly accepted in the diagnosis of the TMJ syndrome continue to be those defined by Schwartz and Laskin.

Objective Clinical Manifestations

Temporomandibular joint pain-dysfunction disorders can be classified into two groups: 1. disorders primarily organic in nature and, 2. functional or non-organic disorders.

The first group comprises the category of TMJ dysfunction or disease proper, that is, disorders directly linked to organic changes or disturbances in the temporomandibular joint and its related structures. These can include displacements of the disk or condyle, inflammatory conditions and arthritis (Lamont-Havers, 1966; Bell, 1969), ankylosis, fractures, muscular dysfunction, and occlusal disharmony (McNeil, Danzig, Farrar, Gelb, Lerman, Moffett, Pertes, Solberg, & Weinberg, 1980). The onset of these conditions may be due to trauma as well as to the adaptive changes, bone resorption and remodeling etc., that occur throughout the life cycle (Blackwood, 1966). A complete classification of the organic conditions which can afflict the temporomandibular joint is found in Bell (1986).

The second category pertains to the disorder commonly termed the "Myofascial Pain-Dysfunction" (MPD) syndrome, whose symptoms are primarily associated with masticatory muscle spasm and unilateral pain (Laskin, 1969). In the case of myofascial pain-dysfunction, there is no evidence of pain originating from organic changes or trauma to the TMJ itself. The distinction between the TMJ/MPD syndromes is, however, still a point of debate in that long-term muscular spasm can produce changes in occlusal harmony as well as degeneration of the temporomandibular joint, while organic or structural changes in the TMJ can also lead to muscular dysfunction. Rather than two separate and distinct entities, the present writer will view TMJ dysfunction and myofascial pain-dysfunction as part of a continuum of polarized but often overlapping clinical signs and symptoms. Often, the distinction between the two syndromes will depend upon the severity and duration of the symptoms, the timing of the examination, and the theoretical orientation of the clinician. Indeed, Bell (1986) indicates that the pendulum of professional opinion swung away from the concept of internal joint derangement with the popularization of the MPD syndrome in the 1970s, only to return to a focus on internal, functional joint mechanics in the 1980s. There are indications, maintains Bell, that a more balanced and moderate approach to temporomandibular disorders, encompassing an integrated perspective on the entire masticatory system, is emerging.

Despite the argument over whether the TMJ syndrome indeed constitutes a true syndrome (Reynolds, 1988), there is a general consensus of opinion over which clinical signs indicate the TMJ pain dysfunction syndrome. These are: 1. pain and tenderness in the TM joints, preauricular areas, and the muscles of mastication, 2. sounds (clicking, popping, crepitus) accompanying condylar movement and, 3. limitation of mandibular movement (Rugh & Solberg, 1979; Ash, 1986). The presence of joint sounds alone is not considered sufficient for determining TMJ dysfunction, nor are psychological factors considered essential.

In addition to the primary signs listed above, the clinician may also include a visual assessment of head and facial symmetry to screen for structural and/or soft tissue abnormalities such as muscular hypertrophy (Schwartz & Chayes, 1966); deviation of the mandible on opening and closing (Greene, Lerman, Sutcher, & Laskin, 1969); mandibular posture and occlusion (Weinberg, 1980); bruxism (Glaros & Rao, 1977); mouth breathing (Garry, 1982); referred pain (Travell, 1960; Campbell, et al., 1982), general postural abnormalities and limitation of movement in the neck, upper and lower back (Gelb, 1977; Gelb & Bernstein, 1983).

Epidemiology and Incidence Rate

The majority of epidemiological studies of mandibular dysfunction have been Scandinavian and have used varying sample sizes representing both patient and general populations (Reider, Martinoff, Wilcox, 1983). In a study of 1069 Swedish shipyard employees, Hansson and Nilner (1975) reported that 79% of the subjects manifested some TMJ or related muscular symptoms, and 30% had a combination of two or more clinical signs. TMJ clicking was the most frequently observed sign, occurring in 65% of the subjects.

In a recent survey of temporomandibular symptoms in a large United States metropolitan area, Duckro et al. (1990) found that the prevalences of self-report symptoms were generally lower than in other studies. Of the 500 subjects interviewed by telephone, 29.8 percent admitted to one or more of the following symptoms: nocturnal bruxing (12.8%), soreness on waking (8.0%), soreness with use (11.8%), joint noise (11.0%), and diurnal clenching (10.8%). The authors also corroborated the observation of Helkimo (1976) that there appear to be no gender differences in the prevalence of temporomandibular symptoms within the general population.

In another recent study, Kleinknecht, Mahoney, and Alexander (1987) also found that more temporomandibular dysfunction symptoms were reported by female than male respondents. The greatest symptom prevalence was found in the age range 30 to 49 years. An important feature of the authors' research is the finding that the TMJ dysfunction symptoms could be empirically clustered into two core groups. Composite I contained five primary symptoms: jaw muscle pain with and without use, joint pain, joint sounds, and bruxism. Composite II contained a variety of symptoms including: ear pain, limited opening, other facial pain, pain in the temple muscles, dizziness, nausea, headache, neck/shoulder pain, ringing ears.

Conversely, in studies of clinical populations, many researchers report that between 65% and 80% of TMJ dysfunction patients are female, with the predominant age group lying in the 20-40 year bracket (Moss & Garrett, 1984). In accounting for this divergence from the epidemiological data, some investigators maintain that women are generally more health conscious than men and tend to seek medical help for pain more readily (Carraro, Caffesse, & Albano, 1969; Agerberg & Carlsson, 1972). Additionally, Reider proposes that women as a group are more sensitive to TMJ pain, sounds, and other symptoms than are men, in spite of the relatively equal distribution of these symptoms in the general population

(Reider et al., 1983). Other authors have suggested that women are more prone to psychosomatic disease (Agerberg & Carlsson, 1972; Heiberg, Heloe, & Krogstad, 1978); and less tolerant of pain (Woodrow, Friedman, Siegelaub, & Collen, 1975). As a result of these findings, as well as the general preponderance of female TMJ patients, most studies of mandibular dysfunction have used primarily female subjects.

In summary, most epidemiological studies of disorders of the masticatory system have concluded that signs and symptoms of mandibular dysfunction occur in a large percentage of the general population, although the incidence of pain or other discomfort warranting medical attention is relatively small. Furthermore, there do not appear to be any sex differences in the distribution of these signs or symptoms. On the other hand, in studies of clinical populations, of those who do seek assistance for problems associated with TMJ dysfunction, female patients are clearly in the majority.

Symptom Mechanisms and Etiological Factors

Pain is the most frequently encountered concern of patients presenting with TMJ pain-dysfunction (Butler, Folke, & Bandt, 1975; Nel, 1978). The pain is usually unilateral in cases of MPD, and commonly bilateral in instances of organic temporomandibular joint dysfunction (Weinberg, 1980). The quality of the pain has been described in varying terms, from a dull aching to a severe, excruciating pain (Weinberg, 1980). One author reports some of his patients relating a feeling of "worms in the bones" and of "teeth growing up into the head" (Alling, 1981).

The quality of pain experienced by the patient is significant in making a proper diagnosis of the particular type of dysfunction. Bell (1982) has categorized orofacial pain into four main groups: a. superficial somatic pain, b. deep somatic and visceral pain, c. neurogenous pain, and d. psychogenic pain. Both superficial and deep somatic pain are the result of noxious stimulation of body tissues containing normal innervation. Pain in this category can reflect involvement of muscles, joints, connective tissue, or the bones; and can be referred as well as localized. Burning or throbbing pain which is aggravated by palpation or movement may indicate inflammation or muscle spasm. Neurogenous pain is characterized by the presence of paroxysmal shooting pains extending along the course of a particular nerve. Psychogenic pain, a subset of the somatoform disorders, primarily involves the complaint of pain in the absence of sufficient pathophysiological or physical findings. The pain does not usually follow the known nerve pathways, and the emergence or exacerbation of symptoms is often related to the onset of psychological conflicts or needs. Secondary gain is present when the pain provides a socially acceptable means of satisfying, or avoiding the psychological needs or conflicts (Waldinger, 1986). The pain experienced by the TMJ pain-dysfunction patient is most often of the deep somatic type involving the masticatory musculature and/or joint structures, although the presence of a psychogenic pain disorder must be considered in some patients.

Etiological theories accounting for the pain mechanisms in mandibular dysfunction can be roughly classified into two groups: a. mechanical displacement theory and, b. muscle dysfunction theories.

Mechanical Displacement Theory

Proponents of the mechanical displacement theory maintain that pain results from condylar displacement due to mandibular overclosure. Loss of posterior occlusion is the primary precipitating factor. Costen's original theory viewed the observed complex of "ear and sinus" symptoms as resulting from pressure applied by the condyle on the auriculotemporal and chorda tympani nerves as well as that transmitted through the temporal bones of the skull (Costen, 1934; Moss & Garrett, 1983). Further anatomical research, however, discounted Costen's nerve compression explanation for facial and head pain (Zimmerman, 1951). Recently, other investigators have proposed that pain may result from posterior and superior condylar displacement impinging upon, and causing inflamation of, the highly vascular and innervated connective tissue of the retrodiscal pad, the posterior portion of the articular disc which attaches to the posterior wall of the joint capsule (Weinberg, 1979a, 1979c; Hylander, 1980; Moss & Garrett, 1984). In a recent research article, Schellhas, Wilkes, and Baker (1989) reported that internal derangement of the TMJ, accompanied by inflammation of the joint tissues, is of particular importance in the origin of facial pain and cephalgia. The pain referral appears to occur through abundant free nerve endings belonging to the auriculotemporal branch of the trigeminal nerve which innervates the posterior joint capsule, or branches of the masseteric and temporal nerves which serve the anterior joint capsule (Kawamura, 1980). Schellhas et al. (1989) used magnetic resonance imaging (MRI) to detect inflammation, presumably responsible for the patients' pain, in joints which were otherwise free of associated mechanical joint signs such as clicking, crepitus, and locking.

From the mechanical displacement point of view, increased activity in the masticatory muscles seldom initiates the pain-dysfunction syndrome, rather it is, a result of disturbed condyle/fossa and occlusal relationships. In such cases, because of improper condylar position or faulty occlusion, the forces of the musculature are not adequately buffered and act directly upon the temporomandibular joint leading to pain and dysfunction (De Boever, 1973). The condition may also be aggravated by the presence of chronic oral habits such as bruxism or continued clenching, gum chewing, pipe smoking, and mouth posture required for playing various musical instruments.

Clicking and other joint sounds accompanying mandibular movement are thought to be due to posterior, medial, or anterior displacement of the articular disc in relation to the condyle. Dislocation of the disc can occur for a variety of reasons including chronic overstrain of the elevator muscles, iatrogenically disturbed function, faulty occlusion, and developmental factors (Wabeke et al., 1989). Functional disorders of the articular disc can also be related to structural abnormalities, or to dislocation or other dysfunction arising out of extrinsic trauma sustained in motor vehicle accidents, fights, or sports injuries (Wabeke et al., 1989). Crepitus and other grating or scraping sounds are usually associated with perforation of the disc and are a sign of advanced joint dysfunction and possible degenerative changes (Weinberg, 1980). Despite the relative prominence given to joint sounds such as clicking, there is no consensus as to their significance in the etiology and long-term progression of TMJ dysfunction, nor as to how they should be treated, if at all (Wabeke, et al., 1989).

Limitations in mouth opening and deviations during mandibular movement are also often interpreted as a result of faulty condyle/disc function, with the disc being displaced anteriorly and wedged between the condyle and the articular eminence thus preventing the normal translatory motion of the joint (Moss & Garrett, 1984). This type of disc displacement can occur bilaterally or unilaterally. In the former instance, bilateral locking or restriction of mandibular opening results; in the latter, mandibular deviation will be apparent. Other explanations for limitation reviewed by Moss and Garrett include organic changes such as ankylosis due to fibrous adhesions or calcifications of the articular surfaces, inflamation of the capsular ligaments, and masticatory muscle spasm.

Muscle Dysfunction Theories

Muscle dysfunction theories of the TMJ pain-dysfunction syndrome maintain that pain results from masticatory muscle hyperactivity and spasm. These fall into two major groups: 1. Neurophysiologic theories and, 2. Psychophysiologic theories.

1. Neurophysiologic theories of the TMJ syndrome postulate abnormal occlusal relationships which lead to altered proprioceptive impulses and inappropriate muscle

activity. Such activity can take the form of increased muscle tonus, spasm, clenching and bruxism (Ramfjord, 1961a, 1961b), and is linked to a background of psychic tension, anxiety, and stress (De Boever, 1973). The excessive muscle activity may lead to pain in the muscles, ligaments, and the temporomandibular joints through abnormal mandibular posture. Other signs and symptoms of dysfunction, such as mandibular deviation and limitation, are due to muscle splinting and spasm which inhibit mandibular movement on the affected side.

2. Psychophysiologic theories of TMJ pain-dysfunction are based on the concept that pain is primarily the result of increased masticatory muscle activity, incoordination, and spasm intimately related to stress response and other psychological factors.

Schwartz and his colleagues were the first to advance the theory that psychological factors such as stress, nervous tension, anxiety, etc. play a key role in the onset, maintenance, and exacerbation of temporomandibular joint pain-dysfunction (Schwartz, 1955, 1956; Schwartz & Cobin, 1957). From this point of view, the patient's response to stress takes precedence over even severe malocclusion in the development of the syndrome (Greene, 1980). On the other hand, the psychophysiologic theory refers to acute malocclusion produced by changes in muscle balance. This symptom develops quickly and usually subsides when the muscular problems are resolved. Occlusal restorations and other equilibration procedures are contraindicated, although they may be necessary if the muscle imbalance is not quickly rectified and the occlusion shifts to restore a proper functional relationship.

The psychophysiologic theory was further developed by Laskin with the theory of the myofascial pain-dysfunction syndrome. As with Schwartz, Laskin maintained that masticatory muscle spasm is the primary cause of pain and the other signs and symptoms of the pain-dysfunction syndrome. Spasm can be initiated in one of three ways: 1. muscular overextension, 2. muscular overcontraction, or 3. muscle fatigue (Laskin, 1969, Mikhail & Rosen, 1980). Laskin (1980, 1986) includes trauma as an etiological factor in muscle spasm and the pain-dysfunction syndrome. The trauma can occur in many ways, among the most common being a blow to the jaw or some other neck or head injury sustained in an accident. The neuromuscular mechanisms involved in mandibular dysfunction related to "whiplash" or similar injuries have been discused by Lader (1983). Other common sources of trauma to the temporomandibular joint complex and associated musculature include dental procedures such as extractions, orthodontics, or restorations.

Pullinger and Monteiro (1988), in a comparison of temporomandibular disorder patients and two other groups, one asymptomatic, and the other displaying mild or early signs of dysfunction, found that a history of trauma, defined as head or neck trauma--as in hyperextension/hyperflexion injuries--orthodontic treatment, or oral surgery, was the most significant factor characterizing the TM disorder group. The specific contribution of these history factors in the onset of temporomandibular dysfunction is often anecdotal and inconclusive. With respect to the role of hyperextension/hyperflexion (whiplash) in mandibular dysfunction, however, Schneider, Zernicke, and Clark (1989) report promising results from their kinematic studies of jaw-head-neck models during computer simulated rear-end collisions.

Although muscle spasm may be initiated by mechanical dental factors such as improper dental restorations, prostheses, malocclusion, and alveolar bone resorption, Laskin believes that most cases of the pain-dysfunction syndrome are due to muscular fatigue produced by stress-related muscular hyperactivity. This hyperactivity can take the form of "chronic parafunctional habits" including clenching and bruxing the teeth, habits he considers to be an "involuntary tension-relieving mechanism in response to psychological stress" (Laskin, 1980).

Clark, Beemsterboer, and Rugh (1981) documented the direct link between pain-dysfunction symptoms and nocturnal muscle activity (bruxism) by monitoring masseter EMG levels in 85 subjects with varying degrees of jaw dysfunction. A significant correlation was found between the amount of nocturnal masseter activity and the clinical manifestations of mandibular dysfunction.

Moss, Ruff, and Sturgis (1984) found that diurnal bruxism was able to discriminate between a group of TMJ pain patients and a no-pain group. Interestingly, the authors found that the habit of resting the hand on the side of the head was negative predictor of the TMJ pain group. On the other hand, Marbach, Lennon, and Dohrewend (1988), reported that TMJ pain dysfunction patients did not differ significantly from non-TMJ controls in a self-report of oral habits. The authors also observe that although the patient group did not self-report habits such as bruxing more than the controls, they had more frequently been alerted to these behaviours by their dentists.

Temporomandibular joint sounds such as clicking and popping are caused by muscle incoordination rather than condylar displacement, while crepitus and other grating sounds reflect actual joint pathology (Laskin, 1980). The muscular incoordination may be occasioned by injury or acute strain, and typically involves the lateral pterygoid (Wabeke et al., 1989). Limitation and deviations in mandibular movement are primarily due to muscle spasm as is the muscle tenderness to palpation characteristic of pain-dysfunction patients.

Although there is general agreement on the cardinal signs of TMJ dysfunction, there is less consensus regarding the relative importance of the various contributing factors in the etiology of the condition. As Mongini (1990) indicates, this disagreement arises for four reasons: 1. different etiologic factors may be operating simultaneously; 2. these etiologic factors may lead to different signs and symptoms in different patients; 3. other simultaneously occurring disorders of the hormonal, vasomotor, and nervous systems may be superimposed over the TMJ-related disorder; and 4. the presence of these other disorders may cause the patient to receive different diagnoses depending upon the specialist consulted.

Psychological Implications

With the psychophysiologic pain-dysfunction theory maintaining that stress, anxiety, and other emotional and personality factors play a significant role in the onset and maintenance of the temporomandibular dysfunction, it is not surprising that the psychology of the TMJ patient has been the subject of many studies. Rugh and Solberg (1976) have reviewed and classified them according to their predominant theoretical orientation. The most significant of these to consider here are: (a) psychoanalytic concepts, (b) personality traits, and (c) learning theory.

Psychoanalytic Concepts

One of the first psychological investigations in this area was conducted by Moulton (1955b), a psychoanalytically oriented psychiatrist and associate of Schwartz. After a psychiatric study of 35 TMJ patients, Moulton concluded that all but four manifested signs of psychological disturbance. Twenty were evidently anxious and nervous, 18 of the 35 either bruxed or clenched their teeth, presumably when repressing emotion, especially anger. Furthermore, eleven of the patients were diagnosed as psychotic or prepsychotic. In another study, Moulton linked the pain-dysfunction syndrome to hysterical conversion reaction of repressed sexual guilt (Moulton, 1955a). The conclusions of Moulton's work suggest that TMJ pain-dysfunction patients tend to exhibit various personality and emotional disorders characterized by increased muscular response to emotional and psychic stress.

Lefer (1966) concluded that the TMJ patient usually has "poor ego boundaries and utilizes bodily reactions to diminish the level of anxiety aroused by the threatened eruption into awareness of oral sadistic, extractive, and incorporative needs" (p. 149). These patients often see their dentists as authority figures with whom they form symbiotic relationships to protect themselves against feelings of bereavement from threatened or actual loss of the mother (Lefer, 1971).

Fine (1971), in a larger study involving 50 TMJ patients and 50 non-TMJ controls, concluded that 76% of the experimental group subjects manifested psychiatric symptoms. The most frequently observed disorders were characterized as depressive-anxiety reactions initiated by bereavement experiences and other emotional stressors. Fine thus concluded that psychological factors play a more important role in the onset and development of the mandibular pain-dysfunction syndrome.

In summary, psychoanalytic concepts hold that symptoms of TMJ dysfunction arise primarily out of denial or repression of anxiety, conflicts, or emotional needs and impulses, as well as past or present experiences involving bereavement, frustration, or guilt.

Personality Traits

Most personality studies of TMJ pain-dsyfunction patients have used structured interviews and/or written personality inventories. The most frequently used assessment instrument has been the Minnesota Multiphasic Personality Inventory (MMPI) which has generally revealed that pain-dysfunction patients tend to have personality profiles similar to those of patients suffering from a wide variety of other psychophysiologic disorders (Olson, 1980). Like these other patients, TMJ pain-dysfunction patients often suffer from related psychophysiologic disorders as reported by Berry (1969) and Lupton (1966).

In one of the earliest studies of the "TMJ personality" using the MMPI, supplemented by the Cornell Medical Index and the Edwards Personality Profile, Kydd (1959) found that 23 of 30 TMJ subjects (76%) evidenced signs of significant emotional disturbance and presented as anxious, tense, apprehensive, and overreacting to pain. McCall, Szmyd, and Ritter (1961) administered the MMPI to 70 TMJ patients and two non-TMJ control groups of 70 subjects each. Following an analysis of the inventory results, it was possible to discriminate between the TMJ and the control groups on the basis of 48 significantly different responses. Of these TMJ specific items, 22 included somatic complaints, while the remainder were associated with chronic anxiety, worry, and other miscellaneous factors. McCall et al. were not, however, able to define a particular personality profile from their data, although they anticipated this would eventually be possible with the development of better research instruments. Other personality studies have produced equally inconclusive results: some investigators report definite TMJ personality characteristics, while others fail to find any group personality tendencies.

In a study of 37 female TMJ patients using the MMPI, Interpersonal Adjective Checklist, and the Thematic Apperception Test (TAT), Lupton (1966, 1969) described the group as rigid and hypernormal. These women were perceived as being matriarchal, dominant, responsible, generous, managerial, and narcissistic; they also seemed to rely heavily upon denial and repression in order to maintain a consistent self-concept.

Similar personality trends were reported by Molin, Schalling, and Edman (1973) who found that, compared with a normal group, the TMJ sample rated higher scores in neuroticism, somatic anxiety, muscular tension, aggression and superego strength. They also had a tendency to be more conscientious, responsible, orderly and serious. Schwartz, Greene, and Laskin (1974) also found that pain-dysfunction patients obtained elevated scores in the "neurotic triad" of the MMPI (the hypochondriasis, depression and hysteria scales). In a multiprofessional Scandinavian study of 113 MPD patients and 46 control subjects, Heloe, Heiberg and Krogstad (1980) concluded that pain-dysfunction patients tend to over control their emotions, particularly aggression. However, both groups scored high on the MMPI Anxiety scale.

On the other hand, Solberg, Flint, and Brantner (1972), also using the MMPI on 29 TMJ patients with an equal sized control group, reported no common personality traits or patterns. Some of the patients demonstrated greater anxiety, but this was viewed as within the range of a normal personality profile.

Schumann, Zwiener, and Nebrich (1988) in an interesting study correlating MMPI characteristics and EMG activity from the temporalis, masseter, and anterior digastric muscles, in 70 TMJ dysfunction patients, found that the majority of patients produced psychosomatic "V" in the MMPI profile. The female patients scored significantly higher than healthy controls in scales 1(Hs), 2(D), 3(Hy), as well as in 6(Pa), 7(Pt), 0(Si), and L.

Two recent studies have used the Symptom Checklist-90-Revised (SCL-90R) with temporomandibular dysfunction patients. Butterworth and Deardorff (1987) reported the emergence of three discrete SCL-90R profile subgroups within a group of 100 successive TMJ dysfunction patients presenting to a craniomandibular treatment centre. The first of the three subgroups was described as "psychologically normal"; the second demonstrated elevations on the somatization, depression, and anxiety scales, and were deemed similar to the "hypochondriasis" MMPI profile. The third subgroup showed significant elevations on scales relating to somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobia, and psychoticism. This subgroup was considered severely distressed and termed the most "psychopathological-appearing." Alpern, Nuelle, and Wharton (1988), using the MMPI, also report finding three TMJ patient subgroups corresponding to the categories described by Butterworth and Deardorff (1987).

Lee and Lee (1989) also reported the use of the SCL-90R with a group of 219 temporomandibular disorder patients in a Korean hospital. In their study, somatization and anxiety emerged as the two most elevated scales. The authors concluded that the likelihood of somatization disorder, generalized anxiety disorder, conversion disorder, and hypochondriasis was significant in the temporomandibular disorder patients. In applying the findings of Lee and Lee (1989) to North American patients, it is important to consider the influence of cultural and social factors on the test results.

In addition to the standard personality instruments, the Social Readjustment Rating Scale (SRRS) has also been used to assess the impact of stress in TMJ pain-dysfunction patients (Stein, Loft, Davis, & Hart, 1982). The TMJ Scale and TMJ Scale Report (Levitt, Lundeen, & McKinney, 1987) is a recently developed instrument which seeks to provide a global assessment of physical signs and symptoms combined with psychosocial and stress factors.

Rugh and Solberg (1979) concluded that there does not appear to be any one personality trait or characteristic common to all TMJ pain-dysfunction patients. Rather, TMJ patients may display a variety of personality characteristics which may or may not have a significant impact on the emergence and manifestation of a TMJ disorder. Past research has focused on identifying personality types within what was considered a generally heterogeneous patient population. Current efforts are seeking to more precisely discriminate between diagnostic subgroups of TMJ dysfunction patients and identify specific personality correlates. Although Rugh and Solberg (1979) considered further inquiry into the TMJ personality to be a futile pursuit, ongoing and future research utilizing more sensitive assessment procedures and instruments may provide additional insight into the psychological dynamics of the TMJ pain-dysfunction patient.

Learning Theory

A learning theory approach to TMJ pain and dysfunction begins with the concept of illness behavior, i.e., the ways in which different types of people perceive and respond to signs and symptoms of illness (Mechanic, 1962). The social and cultural background of the individual exerts an important influence over the variety of behavioral responses given to a specific illness, or its signs and symptoms. These behavioral responses include pain recognition, use of health care services, and the tendency to seek release from normal obligations (Speculand et al., 1983).

From a learning theory perspective, pain behavior, like any other behavior, is learned primarily in a social context as a result of observation and modeling (Craig, 1983). The child observes various modeled behaviors and the consequences that ensue. Those behaviors that are followed by some form of reinforcement will tend to be assumed by the child as socially correct or acceptable. In this way, attitudes toward pain and actual pain behavior itself are transmitted from parent to child (Melzack, 1973). Following this model, some types of TMJ pain-dysfunction can also be viewed as a learned behavior occurring in response to certain stimuli and contingent upon subsequent positive reinforcement.

Fordyce (1976) distinguished between two categories of pain: (a) respondent pain, and (b) operant pain. Although there has not been an extensive analysis of the TMJ pain-dysfunction syndrome from this perspective, the concepts of respondent and operant pain offer important insights into the psychological dimension of the syndrome.

Respondent pain is controlled by specific stimuli and invariably follows presentation of the stimuli. Operant pain, although it may also be a response to antecedent stimuli, is primarily controlled by its consequences. That is, operant pain behavior is maintained by positive or reinforcing consequences. Conversely, operant pain behavior will diminish if not followed by some type of reinforcement (Fordyce, 1976).

It is not realistic to attempt to place TMJ or facial pain exclusively into one or the other category, rather the syndrome may include elements of both respondent and operant pain. The present discussion will deal only with the concept of operant pain and its implications for understanding the TMJ pain-dysfunction syndrome.

The concept of operant pain as it relates to TMJ pain can be viewed in three categories as set out by Fordyce (1976):

1. Direct and positive reinforcement of pain behavior, where the behavior persists because of some continued intrinsic reward or benefit that results. The TMJ pain-dysfunction literature is replete with accounts of patients whose symptoms and complaints dominated their family and personal lives, exhausting family finances and local health services. A careful behavioral analysis of these patients, their families, and the relationship they have with health professionals suggests that there is a great resemblance between TMJ pain-dysfunction sufferers and patients with other common pain syndromes (Rugh & Solberg, 1976; Marbach & Lipton, 1978). The communicative and manipulative aspect of such pain is one of its key reinforcers and operates in the form of increased personal

attention, sympathy, concern, etc. on the part of the patient's significant others (Sternbach, 1974). Other examples of intrinsic reinforcement include medication schedules which use pain as the criterion i.e., "take only as needed" and rest breaks determined by the presence of pain (Fordyce, 1983).

2. Indirect but positive reinforcement of pain behavior by avoidance of aversive consequences, whereby pain excuses the patient from difficult personal, emotional, or social situations. From this perspective, TMJ pain may have, at some point in the patient's history, provided relief or time out from some aversive circumstance. The aversive situation that was avoided in the past does not have to repeat itself to again elicit the same type of pain behavior, any difficult, disagreeable, or otherwise unpleasant situation serves equally well. Thus the pain-dysfunction behavior persists (Fordyce, 1976).

3. Failure of well behavior to receive positive reinforcement; in other words, operant pain behavior is much more likely to develop and continue than is operant well behavior. Fordyce cites the example of an individual of limited talents and skills for whom displays of well behavior imply equivalent displays of well performance and competence. As he is not likely to be very successful in the range of what would be considered normal daily

functioning, this individual receives very little, if any, reinforcement for his efforts. "Such a person," Fordyce continues,

> can be expected to develop an extensive repertoire of behaviors designed both to avoid the aversive consequences of failure and to elicit positive reinforcement from the immediate environment. . . . If practiced long enough, such behavior may lead others to lessen their demands and expectations. . . . The person might have adopted a reclusive, hostile, isolated style designed to keep people (and their performance demands) at a distance. But he might also have developed a vast illness repertoire. Weakness, ease of fatigue, hypersensitivity to pain and stress, and a readiness to get sick in the face of demands may describe much of the behavior repertoire (1974, p. 69).

The learning theory concepts of operant pain behavior described above are only offered as possible psychological mechanisms functioning in the personalities of some TMJ patients. The notion of operant pain behavior cannot be over-generalized to explain all of the TMJ pain-dysfunction syndrome any more than other pain syndromes, but it does provide an interesting and useful perspective on the influence of psychological factors in the development and expression of TMJ and other facial pain.

Treatment

Various types of treatment strategies have been applied to TMJ disorders, from psychotherapy to surgery, and each theoretical perspective on the etiology of the condition necessarily promotes a particular treatment orientation. The present review will consider the principle treatment strategies that have evolved out of the mechanical displacement and muscle dysfunction theories. As with some of the etiological conclusions reached by these two theoretical outlooks, the resulting treatment strategies may overlap, but they do so for different reasons. For the sake of simplicity, the different types of treatment procedures can be placed in two categories: (a) dental approaches, and (b) psychological approaches.

Dental Approaches

Three basic dental treatment approaches are commonly used in the management of TMJ pain-dysfunction syndrome: occlusal splints, equilibration procedures, and prescribed medications (Moss & Garrett, 1984).

Occlusal splints perform various tasks among them: restoring proper condyle/fossa relationships by modifying the position of the mandible, redistributing the nonfunctional forces of bruxing or clenching more evenly over the entire occlusion and its musculature, and relieving muscle tension or hyperactivity by altering proprioceptive signaling (Carraro & Caffesse, 1978; Kawazoe, Kotani, & Yamada, 1980); and increasing the resting length of the elevator muscles, thus reducing their isometric tension (Christensen, 1980). Occlusal splint therapy is often followed by equilibration procedures such as selective grinding, restorations, and orthodontics. The aim of these procedures is to permanently reproduce in the occlusion the stabilizing effects of the previous splint by restoring optimal occlusal and mandibular relationships.

Splint therapy and occlusal equilibration has been widely used, with reasonable success. Some of the inherent disadvantages are, however, the long-term nature of treatment in the case of occlusal splints; and the cost and irreversibility of occlusal equilibration.

In an article reviewing long-term results of TMJ treatment in 151 patients, Mejersjo and Carlsson (1983) reported an 80% success rate using primarily occlusal adjustment, splint therapy, and therapeutic exercises. Greene and Laskin (1983) also reported success rates in the 80% range for various treatment modalities. This contrasts with Beard and Clayton (1980) who found 100% reoccurrence of symptoms in patients after discontinuing splint therapy.

Other studies have also considered the role played by the placebo effect in successful treatment. Greene and

Laskin (1972) found that, after a treatment period of 2-6 weeks, 28 of 71 patients had improved with only the use of a non-occluding placebo splint. In another study Goodman, Greene, and Laskin (1976) reported that 16 of 25 patients (64%) experienced complete or nearly complete remission of their symptoms after having received mock equilibration consisting of selective grinding and "adjustment" of non-occluding tooth surfaces. Clarke (1982) also maintained that the success of occlusal therapies is primarily due to the placebo effect.

Wabeke, Hansson, Hoogstraten, and van der Kuy (1989) conclude that success rates in treating TMJ clicking by means of both reversible and irreversible therapies diminish dramatically as the follow-up period increases. In their survey of published outcome studies, they found that after two to seven years, as much as to 80 percent of patients experienced a resumption of joint clicking. The authors indicate, however, that current treatment philosophy is increasingly oriented to obtaining relief from pain and obvious dysfunction, while accepting that clicking of the joints may remain as a benign symptom.

Commonly prescribed medications used in the treatment of the TMJ pain-dysfunction syndrome include analgesics and antiinflammatory agents, muscle relaxants, steroids, antivertiginous drugs, and vitamins and minerals (Hall, 1982).

In another placebo study, Greene and Laskin (1971) compared the effect of meprobamate, a tranquilizer and muscle relaxant, with a placebo drug. The authors found that 58% of the patients administered meprobamate reported improvement, as did 31% of the placebo patients. Laskin and Greene (1972) also reported an extension of their research in a study which evaluated the influence of the doctor-patient relationship on placebo drug therapy for MPD patients. In this project, 50 MPD patients were prescribed a placebo drug accompanied by precise dosage instructions and assurances of its effectiveness. At the end of the four week experimental period, twenty-six of the fifty patients (52%) reported some improvement in their condition with eight not requiring further treatment.

Psychological Approaches

A number of psychological approaches have been used in treating TMJ patients. From the mid 1950s with the exploration of psychological factors operating in TMJ disorders, psychotherapy in the form of individual counselling or group therapy has been prescribed (Moulton, 1955b; Kydd, 1959; Lefer, 1966; Lupton, 1969; Pomp, 1974; Marbach & Dworkin, 1975; Kopp, 1979). Hypnosis has also been utilized for pain and symptom control (Tarte & Spiegel, 1977), as has acupuncture (Corcos & Brandwein, 1976; Quint, 1982).

Another frequently used therapy procedure stemming from the muscle hyperactivity theory is that of relaxation training, based primarily on Jacobson's Progressive Relaxation technique (Gessel & Alderman, 1971; Reading & Raw, 1976; Raft, Toomey, & Greg, 1979). In the early 1970s biofeedback training began to be used as an adjunct to traditional progressive relaxation therapy. Since that time biofeedback has emerged as one of the foremost psychological therapies in the treatment of TMJ pain-dysfunction disorders. In the following pages the theoretical basis for biofeedback therapy and the extent of its application in the syndrome will be reviewed.

B. Biofeedback and TMJ Pain-dysfunction Disorders

Rationale for Biofeedback Therapy in TMJ Disorders

The relatively recent discovery--in occidental thought at least--that voluntary and involuntary or autonomic physiological processes can be influenced or controlled by conscious volition is fundamental to the concept of biofeedback. Basmajian (1979) described biofeedback as:

> the technique of using equipment (usually electronic) to reveal to human beings some of their internal physiological events, normal and abnormal, in the form of visual and auditory signals in order to teach them to manipulate these otherwise involuntary or unfelt events by manipulating the displayed signals. This technique inserts a person's volition into the gap of an open feedback loop--hence the artificial name biofeedback . . . . (p. 1).

Implicit in the theory of biofeedback is the assertion that any physiological process which can be monitored and meaningfully "fed back" to the individual can be willfully controlled or altered by that individual. In other words, the subject has the capacity to self-regulate physiological processes much more than was traditionally believed. It is this capacity for self-regulation or normalization of physiological process, particularly those which are maladaptive and contributing to illness, which makes biofeedback a treatment approach and philosophy of potentially great importance.

Biofeedback therapies have evolved to occupy a central role in the relatively new field of behavioral medicine. Behavioral medicine directs its knowledge of psychology, learning theory, and behavior therapy toward the treatment of a variety of physical disorders and maladaptive behaviors which cannot be classified as entirely physical or psychological. One of the key points of departure from traditional medicine is the redefinition of the doctor-patient relationship explicit in behavioral medicine. In this new health care model, the patient assumes greater responsibility for the prevention and recovery from illness (Olson & Schwartz, 1987).

One of the most frequent applications of biofeedback is in the treatment of psychophysiologic or psychosomatic disorders. Such disorders are characterized by the complex interplay of psychological factors and physiological responses in the onset or maintenance of the disorder and its clinical signs and symptoms. Tarnopolsky and McLean (1976) describe psychosomatic illness as occurring when "physical predisposition, psychological threat and a psychological dynamic vulnerability complementary to that threat are found in the same individual at the same time" (p. 96). Biofeedback provides a means of regulating or modifying the interaction of the contributing physical and psychological factors with a view to limiting and, ultimately, arresting the illness cycle.

The place of biofeedback in the context of TMJ therapy is most directly seen in relation to the muscle hyperactivity etiological theory of the pain-dysfunction syndrome. As Schwartz (1955, 1956) and Laskin (1969) maintain, the pain syndrome arises primarily out of masticatory muscle dysfunction, primarily consisting of abnormally elevated muscle tension and/or spasm. Jacobson (1967) writes that suffering and pain is physiologically related to increased muscle tension; as the patient relaxes to a near zero level of activity, pain responses diminish or disappear. Jacobson notes, however, that pain does not appear to diminish proportionally with muscle tension, but only when total relaxation is achieved. One of the goals, then, of biofeedback training in the TMJ pain-dysfunction syndrome is to enable patients to reduce their masticatory muscle tension or hyperactivity as much as possible while maintaining general overall relaxation.

While the muscle hyperactivity characteristic of the TMJ pain-dysfunction syndrome can result from mechanical or physical irregularities such as trauma or occlusal imbalances, this theory maintains that muscle hyperactivity is more often due to psychophysiological causes, primarily related to the patient's response to stress, environmental as well as inner psychic stress. These two types of stress can be viewed as (a) physical stress and, (b) psychological stress. Some of the major investigations into the relationship between physical and psychological stress in temporomandibular disorders will be reviewed in the following discussion.

Stress and Temporomandibular Disorders

Haber, Moss, Kuczmierczyk, and Garrett (1983) define physical stress as a state of discomfort resulting directly from some physical activity or event. Psychological stress, on the other hand refers to the discomfort arising out of the subjective interpretation of some event. The two types of stress are often related, and both may be implicated in the pain-dysfunction syndrome.

A number of studies have been published which discuss the link between psychological stress and increased masticatory muscle activity. Copeland (1954) presented 80 case studies of mandibular joint dysfunction in which abnormal muscle tension related to temporary "emotional disturbances or mental anxieties" was a significant etiological factor. On the basis of self-reports and clinical examinations of 900 patients, Franks (1965) determined that emotional causes were clearly implicated in bruxism and mandibular dysfunction. He did not find any significant differences between men and women in the incidence of bruxism.

In another early investigation into the effects of psychological stress on masticatory muscle tension, Perry, Lammie, Main, and Teuscher (1960) submitted eight dental students to an identical stress situation and observed their pre and post stress levels of masseter and temporalis EMG activity. From this study the authors concluded that: 1. Electromyography is useful in determining states of anxiety, and tensions in the muscles of mastication. 2. Immediate stress situations produce increased muscular activity in individuals with probable predisposing long-term emotional stress build-up. 3. Clinical and anatomical evidence exists for the often described tensions and aches within the masticatory musculature of individuals under emotional stress.

In a continuation of the work of Perry et al., Yemm (1968) subjected thirty subjects to two kinds of stressors; one where the subjects were to squeeze a pressure bulb to maintain a predetermined pressure, and the second, where subjects were to respond to a column of randomly flashing lights by pressing the corresponding buttons arranged in a horizontal row. In both tasks speed and accuracy of response were monitored with instantaneous feedback given to the subjects in the case of incorrect or hesitant responses. Yemm found that, in the bulb squeezing test, masseter muscle activity was high before the task, increased yet further during, and subsided to the lowest level after the task. In the flashing light test, peak levels of muscle activity coincided with incorrect responses and presentation of the error signal. Yemm also observed a proportional relationship between the number of mistakes made and the magnitude of the muscle activity. Yemm concluded that masseter and temporalis muscle activity increases under stress.

Similar studies conducted by Yemm (1969a, 1969b, 1969c, 1971) reinforced this conclusion and added the significant observation that while TMJ dysfunction patients responded to stress by increasing masseter muscle activity, unlike normal subjects, their response over the course of the experiment did not diminish. Yemm postulated that pain-dysfunction patients are poor at adapting to stress, whether experimental or everyday; this could lead to continued and prolonged loading of the masticatory system, resulting in eventual dysfunction (1969b).

In a study of the effects of anxiety and frustration on masticatory muscle tension, Thomas, Tiber, and Schireson (1973) induced experimental anxiety and frustration in a group of TMJ patients and in equivalent non-TMJ controls. The authors found that only the TMJ group responded to the experimental anxiety with increased masseter and temporalis muscle activity, however, both groups responded to the frustration condition with the TMJ group demonstrating a significantly higher response than the control group. It appeared that, as a psychological stressor, frustration plays a more important role in the TMJ syndrome than does anxiety.

Mercuri, Olsen, and Laskin (1979) compared changes in EMG activity from the left and right masseter, frontalis, and gastrocnemius muscles, heart rate, and galvanic skin resistance in 20 MPD patients and 20 normal controls during experimentally induced stress. There was a large difference in pretest masseter and frontalis activity between the MPD and control groups, and both groups demonstrated an increase during the stress period. The patient group, however, displayed the greatest increase in masseter EMG activity. Conversely, the control group showed greater EMG activity in the gastrocnemius compared with the patient group. This discrepancy was maintained during the stress period. There were no differences between the two groups on the autonomic measures of skin conductance and heart rate. The authors interpreted theses findings as supporting the concept of response specificity in MPD. In this view, the MPD patient responds selectively to stress with increased EMG activity in the masticatory muscles, but not in other muscle groups.

Gervais (1984) contrasted a group of 24 female subjects with clinical signs and symptoms of TMJ dysfunction and 24 asymptomatic control subjects with respect to temporalis and masseter EMG activity at baseline, and throughout a series of psychological and physical stressors. All experimental stressors produced a statistically significant variation from baseline EMG activity, however, there was no significant difference in stress response between the TMJ and control group subjects. The inconclusive findings of this study may have been due in part to inclusion of subjects within the TMJ group who, in retrospect, manifested symptoms of subclinical severity rather than only those subjects with acute or chronic TMJ disorder.

In a replication and extension of the physical stress test conducted by Yemm (1969c), Rugh and Montgomery (1987) recorded and contrasted masseter EMG, heart rate, galvanic skin response (GSR), digital skin temperature, and respiration rate in 23 temporomandibular (TM) pain patients and a matched control group. Analysis of the EMG recording revealed that baseline masseter EMG was higher in the TM group than in the control group. In contrast to other studies, however, the TM and control groups were not significantly different in their response to the experimental stressor. More precisely, both groups responded similarly to the stress task and did not show differences in their degree of adaptation to the task. The authors concluded that these findings offer partial support to the stress-muscular hyperactivity model of TM disorders.

McGlynn, Bichajian, Tira, Lundeen, Mahan, and Nicholas (1989) crossed occlusal interference and stress variables in an experiment designed to investigate the effect of psychological stress and the presence of an occlusal interference on bilateral masseter EMG activity. The authors recorded masseter EMG activity in eight non-clinical subjects on two separate occasions, before, during, and after viewing horrific and idyllic film clips. During the viewing and EMG recording the subjects wore one of two appliances. One appliance was designed to provide an occlusal interference, and the other provided a non-interfering control condition. The appliance was alternated on the second day of the experiment. The authors reported three principle findings: 1. EMG activity contralateral to the appliance was higher than ipsilateral EMG across all variables, 2. EMG activity increased during presentation of the film clips regardless of content, 3. The change in EMG activity between baseline and the viewing periods was greater with the control appliance.

In summary, the literature generally supports the hypothesized link between stress and increased masticatory muscle activity. The magnitude of the EMG activity in response to the stress is greater in patients than non-patients, and the patients typically demonstrate less adaptability to the stress situation (Dahlström, 1989).

In addition to the research on the relationship between stress and masticatory muscle activity, a number of other investigators have also studied the correlation between psychological or social stress and pain in temporomandibular disorder patients. Some of the more recent reports will be reviewed below.

In a study of the relationship between social stress and pain in MPD patients, Moody, Calhoun, Okeson, and Kemper (1981) contrasted 52 patients with an equal size non-MPD control group. The authors used a measure of perceived social stress rather than experimentally induced stress. The pain assessment consisted of the sum of palpation pain rating from the temporalis, masseter, lateral pterygoid, and posterior neck muscles. As a group, the MPD patients demonstrated both higher levels of subjective stress and higher pain scores than did the control group. There was no significant correlation between pain and subjective stress in either group. The authors concluded that MPD patients do indeed experience greater pain and stress that an asymptomatic control group.

In another study, Moody, Kemper, Okeson, Calhoun, and Packer (1982) obtained a measure of recent life changes in

19 MPD patients presenting for treatment for the first time. All patients reported facial pain of at least one year duration. Two life-change unit (LCU) totals were assessed, one for the time period 1 to 6 months prior to presentation for treatment, and the second for the period 7 to 12 months prior to presentation. The mean LCU total score was greater for the period up to 6 months before treatment than in the 7 to 12 month period. Furthermore, the LCU totals for the MPD patients were higher than the LCU totals reported in other studies of patients with other illnesses. The authors suggest that an assessment of life-change stress may be useful in planning treatment for MPD patients.

The Derogatis Stress Profile (DSP) was used by Lundeen, Sturdevant, and George (1987) to characterize two groups of craniomandibular disorder patients. One group of patients (N=28) was diagnosed with only joint pain, and the other with only muscle pain (N=24). The authors found that both groups of patients did not manifest abnormally high stress levels on the DSP, although the muscle pain group showed a tendency toward higher overall stress scores than did the joint pain group.

In a subsequent study testing the concept of symptom progression from stress-related muscle pain, to combined joint pain, and finally, joint pain alone, Lundeen, George, and Sturdevant (1988) used the DSP to examine the role of stress in three groups of patients within these diagnostic categories. It was found that the combined pain (N=39) and the muscle pain (N=24) groups had equivalent levels of pain intensity and impairment. However, the combined pain group rated lower than the muscle pain or joint pain groups in terms of the DSP global stress rating. The authors concluded that the findings do not support the concept of stress-related symptom progression from muscle pain through to joint pain.

Support for the physical stress concept comes from the studies of Christensen (1971, 1975, 1979, 1981a, 1981b). Christensen reported that experimental maximum clenching and grinding in normal subjects produced pain similar in location and intensity to that experienced by MPD patients. The other signs of dysfunction--tenderness to palpation, joint sounds, limitation of movement--were not found.

Clark, Jow, and Lee (1989) used ten healthy male subjects in an experiment measuring the long-term effects of maximum sustained jaw clenching. During the eighty minute experiment, subjects were instructed to clench with maximum force and hold the contraction to their pain tolerance limit. Masseter and anterior temporalis tenderness was assessed at pretest, during the experiment, and one, two, three, and seven days posttest. The authors reported a significant increase in muscle pain during the experiment, but found that the pain level quickly subsided and no significant residual pain was noted at the time of the follow-up examinations up to seven days later. The findings of this study challenge the concept that pain induced by sustained clenching in healthy normal subjects can be used as a model for chronic jaw muscle pain.

In a related study, Watkinson (1988) used EMG biofeedback to increase the amount of EMG activity during maximum voluntary clenching to the point of pain. In contrast to first trial means without feedback, the maximum clenching with concomitant feedback resulted in a 36% mean increase in EMG activity beyond the previous pain threshold. The authors concluded that the use of EMG biofeedback during maximum clenching allowed the subjects to override central factors, such as pain, which normally protect the muscle and its attachments from potentially damaging overload.

Biofeedback in Temporomandibular Disorders

The work of Perry et al., Yemm, and Christensen provides a basis for the application of biofeedback in the treatment of the TMJ pain-dysfunction syndrome. With the demonstration that pain-dysfunction patients typically exhibit masticatory muscle hyperactivity which is implicated in the development of pain and dysfunction symptoms, and that part of the overactivity may be related to a stress response specific to the mandibular dysfunction patient, the theoretical groundwork was laid for comprehensive biofeedback therapy.

The role of biofeedback therapy in temporomandibular disorders can be summarized as follows: 1. Biofeedback serves to monitor and reduce overall masticatory muscle tension and correct muscular imbalances due to chronic oral habits. 2. Feedback can make the patient aware of the role his own response to stress plays in initiating and maintaining dysfunctional muscle activity.

The use of biofeedback in dentistry is a relatively recent development. Over the last twenty years biofeedback has gone from occupying a tentative research role to actual routine application in the treatment of various stress related dental disorders. Two excellent multidisciplinary works (Gelb, 1977; Morgan, House, Hall, & Vamvas, 1982) have devoted entire chapters to the application of biofeedback in the treatment of TMJ disorders. While not yet extensive, a substantial body of research and clinical literature on the use of biofeedback in temporomandibular disorders has accumulated. The status of biofeedback as a legitimate adjuvant treatment for mandibular dysfunction and pain has been emphasized by the inclusion of an individual chapter on this specific treatment application in an important review of clinical efficacy in biofeedback training (Hatch, Fisher, & Rugh, 1987). The following discussion will review the key developments in the application of biofeedback in temporomandibular disorders.

Solberg and Rugh (1972) published one of the first reports describing a clinical application of biofeedback in dentistry. In this study, fifteen TMJ patients were given small EMG units to wear during the day. These units continuously monitored masseter muscle activity and whenever it exceeded a preset threshold level, a warning tone was emitted by the unit. The patients found that they typically clenched or bruxed their teeth when confronted with stressful situations. As a result of the self-monitoring the patients were able to identify such situations, avoid them, or find other ways of coping with the stress. Upon completion of the study, ten of the fifteen patients reported a significant improvement in their condition.

Mulhall and Todd (1975) reported the case of a 32 year old male patient who also was supplied with a portable EMG unit. The unit was set with a variable sensitivity threshold which was gradually adjusted in order to progressively shape the patient's awareness and frequency of clenching. After six weeks of self-monitoring, the patient returned the unit claiming an improvement in symptoms.

In another important research project, Budzinski and

Stoyva (1973) compared the effects of visual feedback, auditory feedback, irrelevant feedback, and no feedback in reducing masseter muscle tension in eighty subjects. Although all participants were given written instructions to relax all muscles as much as possible, significant reduction in masseter EMG was observed only in the meaningful feedback groups. The authors concluded that biofeedback training is a potentially useful therapy in the treatment of bruxism and TMJ dysfunction.

Carlsson, Gale, and Ohman (1975) reported one of the first clinical applications of biofeedback in the treatment of TMJ dysfunction. The patient was a twenty-one year old woman described as having long-term pain in the TMJ, right lower jaw, and the masseter. After 18 sessions of weekly, or bi-weekly masseter biofeedback training, the patient was able to control her masseter muscle tension and pain. At time of writing, the authors reported that the patient had been totally pain-free for six months.

In a similar case report, Carlsson and Gale (1976) reviewed the progress of a 59 year old woman whose primary complaint was severe pain in the left masseter. In baseline recordings obtained during relaxation, the left masseter showed more than five times the activity of the right, as well as spontaneous, unilateral contractions. After nine weekly biofeedback sessions the patient reported a significant relief in pain, with no relapse after a one-year period.

Gessel (1975) conducted a large-scale study using biofeedback as a treatment for facial pain. In this study, 23 MPD patients were given temporalis and masseter feedback for at least six thirty minute sessions and required to practice at home for one half hour daily. Those who did not improve after six sessions were considered failed and transferred to the second phase of the experiment, in which an anti-depressant drug program was initiated. Of the 23 patients, 15 reported satisfactory improvement of their symptoms after an average of five biofeedback sessions. On the basis of the pretreatment interview in which each of the participants had been rated in terms of high social drive, covert and overt depression, and social disability, Gessel found that the depression indicators discriminated between the patients responsive to biofeedback and those who derived no benefit from the training. Those who failed to respond to either treatment manifested signs of severe depression.

In another study using 24 MPD patients (Dohrmann & Laskin, 1976; 1978), 16 experimental subjects received nine sessions of auditory feedback from the masseter muscle. The remaining eight patients were assigned to a control group and told they would be receiving muscle relaxing electrical stimulation from the electrodes. At the end of the ninth session only two patients in the experimental group still complained of pain, as compared to four in the control group. Additionally, the experimental group showed a mean increase of 8.5mm in maximum opening without discomfort, compared to 2mm in the control group. The percentage of patients experiencing muscle tenderness upon palpation also decreased significantly in the experimental group (from 81% to 43%), but not in the control group (80% to 75%). At a twelve month follow-up, 12 of the 16 experimental patients claimed to be symptom free. The authors concluded that biofeedback is effective in treating the MPD syndrome.

Berry and Wilmot (1977) reported the results of a clinical trial of biofeedback with 35 mandibular pain-dysfunction patients. Each of the patients received an average of three biofeedback sessions. Although the authors did not state the length or frequency of the training sessions, 61% of the patients were relieved of symptoms within six months, and 85% within a year. One of the major benefits of the biofeedback, in the authors' view, was the increased awareness of masticatory muscle tension reported by all of the participating patients.

Another clinical application reported by Clarke and Kardachi (1977) used nocturnal biofeedback supplied by a portable EMG unit to modify seven patients' bruxism. The feedback signal did not awaken the subjects, but apparently served to rouse them to a lighter stage of sleep. During the treatment period, patients used the biofeedback unit nightly for one to four weeks after which reduction of symptoms was reported. The only exception was the seventh patient who used the EMG unit irregularly due to the inconvenience involved. This patient did not derive relief from either biofeedback or other forms of therapy. The authors concluded that biofeedback used in conjunction with some form of counselling or other psychotherapy is an appropriate form of treatment for the pain-dysfunction syndrome.

Olson (1977) described a study in which 15 MPD patients who had proven non-responsive to drug and splint therapy were randomly divided into three groups. The first group (N=5) received masseter EMG feedback from the painful side, the second group (N=4) received frontalis feedback, and the third group (N=6) was given both frontalis feedback and psychotherapy. The participants attended twelve biofeedback sessions and most attained a low level of muscle activity within the first six sessions. On comparing pre and post-treatment symptom evaluations, Olson found that the first group had benefitted from a slight decrease in pain but not in muscle tenderness, joint sounds or limitation; and one patient in the second group experienced complete remission, but there was no change in the other patients. Two patients in the third group reported complete remission of symptoms, three patients had little or no pain, and one was unchanged. Olson offered three conclusions: 1. The combination of biofeedback and psychotherapy is more effective than biofeedback alone; 2. Biofeedback is not as effective with patients non-responsive to splint and drug therapy; and 3. Feedback from the masseter may be more effective than from the frontalis.

Peck and Kraft (1977) reported a clinical trial of biofeedback training with 18 tension headache, eight back and shoulder pain, and six female TMJ pain patients. All the subjects were given two half-hour biofeedback sessions per week. Although EMG levels dropped across all groups, there was a reduction in pain only in the headache group; the back and TMJ groups did not appear to benefit from the biofeedback. The authors did not indicate the type and severity of clinical signs in the TMJ patients, nor did they report the amount of medication being taken, although this was monitored throughout. Finally, there was no mention made of any type of concurrent home relaxation practice during the period of training. Although the subject sample was small, the authors concluded that biofeedback training is not as effective in the treatment of TMJ and back pain as for tension headache.

Manns, Miralles, and Adrian (1981) combined audiostimulation and EMG biofeedback in the treatment of thirty-three bruxism and MPD patients. The subjects were classified into two groups: The first group (N=14) had a symptom duration of one year or less; the second group (N=19) had symptoms of more than one year duration. Audiostimulation and EMG biofeedback was provided five times per week for an average of 14 sessions. Each session consisted of 15 minutes of audiostimulation, 15 minutes of EMG biofeedback, and 15 minutes of simultaneous audiostimulation and biofeedback. Patients were asked to spend one half hour daily practicing the relaxation exercises used in the sessions. According to clinical assessments carried out before, during, and after the treatment, there was a gradual decrease in symptoms over the treatment period, accompanied by a concomitant reduction in resting EMG levels. Manns et al. concluded that EMG biofeedback and audiostimulation are effective treatment modalities in the myofascial pain-dysfunction syndrome.

Dahlström, Carlsson, Gale, and Jansson (1984) contrasted the outcome of biofeedback training from the masseter and frontalis muscles in 10 acute and 10 chronic mandibular pain patients. The majority of patients reported unilateral jaw pain, and the masseter feedback was provided from the painful side. All patients received six biofeedback training sessions, however, they were not informed as to which muscle site was generating the feedback signal. At the conclusion of the study, the authors found a significant improvement in clinical and subjective mandibular dysfunction symptoms for both patient groups, although there were no significant differences clinically or electromyographically between the two patient groups, regardless of training site. Furthermore, although both groups improved, no significant correlation was obtained between the decrease in clinical symptoms and EMG activity.

In an interesting study Dahlström, Carlsson, Gale, and Jansson (1985) compared the response of 20 mandibular dysfunction patients and 20 healthy control subjects to experimental stress, before and after six sessions of EMG biofeedback training. Bilateral masseter and frontalis EMG activity was recorded during the experimental stress and biofeedback sessions. Both groups of subjects demonstrated significantly greater EMG activity during the experimental stressors than at baseline, however, the patient group EMG was significantly more elevated overall than the control group EMG activity. Following the biofeedback training, the patient group registered significantly reduced EMG activity, whereas the control group EMG was essentially unchanged. Posttreatment EMG levels were not significantly different between groups.

There were also no significant differences in EMG activity between the pain and non-pain sides in the patient group. Significant reductions in clinical dysfunction scores were noted following biofeedback training. The authors concluded that EMG biofeedback training enabled the patients to control their masticatory muscle activity and reduce their reactivity to stress. This decreased reactivity to stress was associated with a reduction in symptoms, suggesting a link between the two factors.

In a recent study, Dalen, Ellersten, Espelid, and Gronningsaeter (1986) compared the effect of EMG biofeedback training in a group of 10 MPD patients and a control group of 9 patients. The study was designed to measure only the specific effect of biofeedback, hence, no relaxation skills were taught. After eight sessions of twice weekly pattern biofeedback combining the EMG activity from the masseters and frontalis muscles, the experimental group showed no significant reduction in masseter EMG, as compared to the pretreatment baseline, nor were they significantly different from the no-treatment control group. The groups were, however, significantly different with respect to pre and posttreatment frontalis EMG activity. There was a reduction in pain intensity in both groups immediately after treatment, and this continued through a six-month follow-up period for the experimental group but not for the control group. The authors concluded that EMG biofeedback from the frontalis and masseter muscles resulted in long-term subjective improvement, and this modality can be recommended in MPD treatment.

The majority of biofeedback applications in the treatment of TMJ dysfunction have used EMG, although there is one report of Galvanic Skin Response (GSR) as the chosen modality. Gross (1975) used a combination of Jacobson's progressive relaxation and GSR feedback to train twenty MPD patients in general relaxation and anxiety reduction. Four months after the initial four-week training period involving one session per week, Gross reported a 90% improvement in function and symptoms. As well, the patients were notably less anxious and dealt better with stress.

In an interesting article describing the outcome of combining biofeedback and cognitive-behavioral psychotherapy, Stenn, Mothersill, and Brooke (1979), provided 11 MPD patients with eight weekly half-hour sessions of progressive relaxation; six of these were given auditory feedback from the masseter throughout the relaxation period, the remaining five were simply monitored for masseter muscle tension. Following the relaxation period, all the subjects met individually with a psychologist for cognitive therapy sessions focusing on the patient's pain response. Over the course of the treatment period, both EMG and pain levels dropped, however, only the subjects receiving feedback showed a significant reduction in pain, whereas there was no significant difference between groups in terms of EMG levels. The authors concluded that the reduction in pain could not be entirely due to the decreased masseter muscle tension, but must somehow be linked to the greater perception of control and competence resulting from the biofeedback training.

Not many studies have appeared which compare biofeedback therapy with other forms of conservative treatment for TMJ dysfunction. Dahlström, Carlsson, and Carlsson (1982) reported the results of biofeedback and occlusal splint therapy in a group of 30 female mandibular dysfunction patients. Fifteen of these subjects received up to six half-hour biofeedback sessions, the other 15 used a full coverage occlusal splint for a six week period. One month after the completion of the study the patients were re-assessed and both groups showed significant improvement in clinical signs and symptoms although there was no significant difference between groups. The authors concluded that biofeedback and splint therapy are equally effective treatment modalities for mandibular dysfunction pain.

In a recent innovative study, Hijzen, Slangen, & van Houweligen, (1986) trained a group of 16 MPD patients to meet and maintain specific levels of masseter EMG activity from the affected side. The criterion levels ranged from 3, 8, 10, 18, 35, and 50 microvolts. EMG biofeedback was provided twice weekly for five weeks. Pre and posttreatment EMG levels during criterion matching trials, pain and dysfunction scores, and patient self-report were obtained from the biofeedback group, a no-treatment control group, and an occlusal splint group. The biofeedback group demonstrated significantly greater improvement in clinical dysfunction scores and subjective symptoms than did the splint or control groups. Furthermore, the outcome of the splint group was not significantly different from that of the control group. The authors suggested that increase in perceived control is the primary treatment effect underlying the effectiveness of biofeedback training.

In a study comparing the effectiveness of biofeedback-assisted relaxation training, Brooke and Stenn (1983) found that relaxation training appeared to be as effective as other forms of conservative MPD treatment. No benefit was apparent when biofeedback was added to relaxation, nor did there appear to be a significant difference in long-term treatment outcome when the relaxation and biofeedback-relaxation patients were compared. Conversely, in comparison with ultrasound and occlusal splint therapy, both relaxation and biofeedback-assisted relaxation therapy were more effective at the six month follow-up. When ultrasound and splint therapy were combined with relaxation training, there was an equalization of treatment outcome, but no significant difference between the various groups. Moss, Wedding, and Sanders (1983) also reported similar results in a study comparing relaxation training and masseter EMG feedback.

Klonoff and Janata (1986) present the results of a single case study using bilateral masseter equalization EMG biofeedback training. In the authors' approach, standard biofeedback relaxation training proved ineffective in substantially altering the patient's pain. They then modified the training protocol with the instruction to balance the EMG activity from both masseters before attempting simultaneous relaxation of both sides. During the six sessions using the equalization approach, the patient reported a substantial concurrent decrease in reported pain which was maintained through a two month follow-up period. The authors conclude that biofeedback equalization training offers important benefits in the treatment of temporomandibular disorders.

Gervais, Fitzsimmons, and Thomas (1989), in a comparison of asymptomatic, subclinical, and frank TMJ pain-dysfunction patients found that the patient group manifested significantly higher EMG activity than the asymptomatic and subclinical groups. Furthermore, the temporalis, more often than the masseters, were found to be the sites of greatest EMG activity. As in Klonoff and Janata (1986), the presence of frequently observed imbalances, often contralateral, between the temporalis and masseters was noted, as was the authors' use of an equalization approach to biofeedback training. The authors concluded that multi-site EMG biofeedback is potentially useful in detecting imbalances and training the patient to restore normal equilibrium in the masticatory muscles.

C. Summary

Masticatory muscle hyperactivity related to individual response to psychophysiological stress has been implicated as a primary etiological factor in the onset and maintenance of TMJ pain-dysfunction disorders. From this theoretical position, a number of psychological, behaviorally oriented therapies have been proposed, the most common being biofeedback and relaxation training. The application of these treatment modalities is based on the psychophysiologic concept of mandibular dysfunction which maintains that a reduction in overall muscle activity, specifically within the masticatory musculature, will result in a decrease of pain and other muscular-related symptoms. Furthermore, training in biofeedback techniques improves patients' ability to self-regulate inappropriate, and often excessive, muscular responses to daily stress, thus breaking the pain-dysfunction cycle.

Although the literature generally agrees that biofeedback training is potentially effective in reducing pain and other dysfunction symptoms in temporomandibular disorders, there is no incontrovertible experimental evidence to support the effectiveness of this application. As Mealiea and McGlynn (1987) indicate, this lack of clarity regarding the effectiveness of biofeedback reflects weaknesses in research methodology, and not inherent weaknesses in the power of biofeedback. Future research should be oriented in a number of directions, one of which is the accurate assessment of clinical outcome following different treatment approaches. More investigation into the relationship between the physiologic variables targeted in biofeedback training and pain and dysfunction symptoms is also needed. As these relationships become clearer, more knowledge of the specificity of effect in biofeedback therapy will be obtained.

Most of the research reported in the literature to date has used small sample sizes or single case designs, usually without adequate control conditions, in evaluating the efficacy of the biofeedback in temporomandibular disorders. Although Mealiea and McGlynn

(1987) correctly indicate that traditional empirical research methodology requires often prohibitively large sample sizes, the single case designs or small sample sizes used in previous research studies has limited the generalizability of their findings and conclusions, particularly in those studies which did not incorporate a patient control group. Clinical or experimental studies based upon more rigorous research designs, using larger sample sizes and appropriate control groups, provide one approach to increasing the validity and generalizability of the findings.

Many of the clinical and research reports have used biofeedback training in combination with some form of concurrent relaxation or cognitive therapy. Isolating the effects of different therapies by incorporating a multi-group design should help determine the relative contribution of each therapy to the outcome of treatment. Finally, the use of more than one standard biofeedback protocol should help clarify the specificity of effect responsible for the success of biofeedback training in temporomandibular disorders. There is also the need to explore the effectiveness of alternate training sites, simultaneous multiple site versus single site training, and training to both balance and reduce EMG activity.

D. Research Questions

The present investigation was conducted to answer the following questions:

1. Is EMG biofeedback more effective than relaxation training or no treatment in reducing the signs and symptoms of temporomandibular pain-dysfunction?

2. Is the biofeedback training protocol more effective when presented according to a daily schedule as opposed to once weekly?

3. What is the correlation between the observed clinical signs and symptoms of mandibular dysfunction and EMG activity?

4. What is the correlation between subjective self-report pain and EMG activity?

5. What is the correlation between EMG activity and subjective percentage improvement?

6. What is the correlation between MMPI scale T scores and EMG activity, clinically assessed temporomandibular pain and dysfunction, self-report pain, and subjective percentage improvement following treatment?

III. METHOD

A. Subjects

The subjects for this study were female temporomandibular joint pain-dysfunction patients referred by a number of dentists in Edmonton, Alberta, and the surrounding area. The participating dentists were sent an introductory letter outlining the study and specifying the criteria for selecting appropriate patients (see Appendix A). The first requirement was that subjects be female TMJ pain-dysfunction patients whose condition be primarily due to neuromuscular factors, rather than degenerative changes or mechanical derangement of the TMJs. The second criterion specified that patients not be receiving another form of TMJ therapy, or that such therapy have proved ineffectual or have stabilized the condition.

Approximately 60% of the referrals came from the practice of Dr. Norman Thomas, a specialist in the treatment of TMJ disorders. The remainder of the patients were referred by other dentists, and a number were directed to the study after hearing a radio announcement (see Appendix B). Of the 109 patients who entered the study, 9 withdrew or failed to complete the treatment program satisfactorily. Approximately 30 other patients were referred or enquired about the study but declined to participate.

Prior to being admitted into the treatment study, the remaining 100 patients received pretreatment TMJ examinations from the referring dentist. In these standardized clinical examinations, occlusion, joint sounds, and palpation pain ratings were obtained from the TMJs and associated musculature. The pain and noise scores were recorded specifically for each site and later summed to provide cumulative palpation pain and joint noise scores. Following the treatment program, the clinical examination was repeated. The participating dentists were kept blind as to the treatment each patient received until after the posttreatment clinical examination had been completed. The clinical examination form used by the dentists is reproduced in Appendix C.

At the introductory biofeedback assessment appointment all potential subjects were provided with an overview of the study (see Appendix D), and an opportunity to raise any questions or concerns. Those who wished to continue in the study were assigned sequentially to one of four groups: weekly biofeedback, daily biofeedback, relaxation, and control. Each subject executed a treatment agreement listing the requirements of the study and paid a $70.00 deposit, to be refunded upon satisfactory completion of the treatment program (see Appendix E).

Once admitted into the treatment program, all subjects completed a TMJ screening questionnaire (see Appendix F) and received an initial electromyographic (EMG) assessment of the temporalis and masseter musculature during resting baseline and stress conditions. They were also administered a battery of psychological tests including the Minnesota Multiphasic Personality Inventory (MMPI), and the Myers-Briggs Type Indicator (MBTI) in order to explore the pretreatment personality characteristics of the patient sample, and whether their treatment outcome could be predicted on the basis of these characteristics. At the conclusion of the initial assessment appointment the treatment and follow-up appointments were scheduled and the subjects were instructed to begin baseline pain symptom charting. The symptom chart used a six-point scale ranging from 0 (no pain) to 5 (severe, incapacitating pain). The subjective pain rating was done four times daily for the duration of the treatment study, at breakfast, noon, supper, and bedtime (Richardson, McGrath, Cunningham, & Humphreys, 1983). These times were selected to minimize disruption and facilitate compliance. The pain symptom rating scale is reproduced in Appendix G.

In summary, the four treatment groups, weekly biofeedback, daily biofeedback, relaxation, and control, were derived from 100 female TMJ pain-dysfunction patients referred to the study by their dentists. All subjects manifested signs and symptoms of TMJ pain and dysfunction which were rated pre and posttreatment on a common rating scale by the participating dentists. In total, 109 patients entered the study and 9 either withdrew or were excluded because of unsuitability or non-completion of the treatment program.

B. Research Design

The treatment program was conducted from September 1988 to April 1990 along an ABA format. The A component consisted of 4 (conditions) by 4 (groups) repeated measures of pre and posttreatment EMG activity at resting baseline and during three stressors. The treatment component B consisted of five 1 hour biofeedback training sessions, regular home progressive relaxation practice, and symptom charting for the weekly and daily biofeedback groups; symptom charting and home relaxation practice only for the relaxation group. The control group received no treatment but charted their pain symptoms for the five week period. For each group, the symptom charting began the day of the initial EMG baseline assessment and continued four times daily until the day of the posttreatment EMG baseline and stressor recordings. The purpose of the relaxation only group was to control for the effect of relaxation practice in the weekly and daily biofeedback groups. The five-week control group period was intended to control for non-specific variables such as spontaneous remission of symptoms or natural healing and resolution of acute symptoms over time. A summary of the research design is presented in Figure 1.

Figure 1

Research Design

Group	Week					
	1	2	3	4	5	6
Weekly	A	B	B	B	B	B A
Daily	A	X	X	X	X	B A
Relaxation	A	B	B	B	B	B A
Control	A	X	X	X	A	

A: EMG resting baseline and three stressors.
B: Five 1 hour biofeedback training sessions, home relaxation practice (weekly and daily groups only). Relaxation group: home relaxation practice only.
X: Symptom charting only.

Each subject's mean EMG microvolt activity from the right and left temporalis and masseter muscles was independently integrated and recorded 36 times for each of the four steps of the pre and posttreatment baseline and stress profile as well as for 5 training trials within each of the subsequent biofeedback sessions. From these mean integrated EMG values, the maximum, minimum, average, and standard deviation were automatically calculated by the software program for each of the four muscles monitored at each step in the baseline and stress profile, and for each of the training periods. Hence, the subjects' reaction during the stress profile and their response in each training period was represented by four separate measures of EMG activity for each muscle site. The muscle placement sites, channel assignment, and session format for the pre and posttreatment recording and training sessions are summarized in Figures 2.1, and 2.2.

Figure 2.1

Pre and Posttreatment Baseline and Stressors Format

EMG Data 1.1-1.4/6.5-6.8	L. Temp. (Ch. 1)	L. Mass. (Ch. 2)	R. Temp. (Ch. 3)	R. Mass. (Ch. 4)
1.1 Resting Baseline	EMG*	EMG	EMG	EMG
1.2 Serial 7 Subtraction	EMG	EMG	EMG	EMG
1.3 Physical Exertion	EMG	EMG	EMG	EMG
1.4 Post Mastication	EMG	EMG	EMG	EMG

*EMG presented as Maximum (MX), Minimum (MN), Average (AV), Standard Deviation (SD).

Figure 2.2

Biofeedback Training Session Format

EMG Data 2.1-6.5	L. Temp. (Ch. 1)	L. Mass. (Ch. 2)	R. Temp. (Ch. 3)	R. Mass. (Ch. 4)
2.1 Resting Baseline	EMG*	EMG	EMG	EMG
2.2 Training	EMG	EMG	EMG	EMG
2.3 Training	EMG	EMG	EMG	EMG
2.4 Training	EMG	EMG	EMG	EMG
2.5 Training	EMG	EMG	EMG	EMG

*EMG presented as Maximum (MX), Minimum (MN), Average (AV), Standard Deviation (SD).

The primary statistical analysis dealt with the various measures of treatment outcome including the EMG data, the pre and posttreatment clinical examinations, the posttreatment questionnaire percentage subjective improvement, and the daily symptom chart ratings.

The EMG data were subjected to three levels of analysis using MANOVA procedures to test for significant differences in mean EMG activity. The first level of analysis contrasted the EMG activity at baseline and during the stressors at the pre and posttreatment stages. The intent was to determine whether or not the subjects responded to experimental stress with increased masticatory muscle activity. The second level of analysis using MANOVA involved contrasts between the pre and posttreatment baseline and stressor EMG means, with a view to determining if there was a significant variation in stress induced EMG activity following treatment. In order to determine whether the four experimental groups differed significantly in their response to treatment, the third level of the multivariate analysis of variance provided between group contrasts at pre and posttreatment, as well as over time.

Multivariate procedures were also used to test for significant within and between groups changes in the pre and posttreatment clinical exams, and daily pain rating. The subjective percentage improvement ratings from the posttreatment questionnaire administered to the treatment groups were also contrasted for between groups differences.

In the second stage of the statistical analysis, Pearson product correlations were calculated between the EMG data and the TMJ symptom questionnaire, the pre and posttreatment clinical examinations, the pain rating scales, and the MMPI. Correlations between the MMPI scores, subjective and objective outcome measures were also obtained. Other questions related to the influence of litigation, and TMJ problems due to injury were also examined. A Chi square analysis of the observed distribution of MBTI types contrasted with the expected female base rate frequency distribution was also conducted.

C. Apparatus and Facilities

The EMG data were collected simultaneously from the masseter and temporalis muscles using the four channels of a Biocomp 2001 biofeedback system and stored on 5.25-inch floppy diskettes by the accompanying Apple II microcomputer. The raw electromyographic activity was registered from the 80 to 400 Hz bandwidth and integrated every 2 to 10 seconds depending upon the length of the recording or biofeedback training period. Summary statistics, including maximum, minimum, mean, and standard deviation for each muscle site, were automatically calculated by the software program following each recording or training period.

Four sets of reusable J & J SE-40 silver/silver chloride electrodes were used, one for each muscle group. These were attached to the subject using Hewlett-Packard double-sided 8 millimeter opening adhesive collars and Hewlett-Packard Redux Creme electrolyte. The muscle site and channel assignments were standardized as follows: channel1: left temporalis, channel 2: left masseter, channel 3: right temporalis, channel 4: right masseter. The biofeedback equipment was returned to the manufacturer for calibration at the outset of the study. The biofeedback assessment and training sessions took place in a quiet treatment room in the author's private practice facility. All subjects were seated upright in a comfortable reclining chair, and the room lights were dimmed.

D. Procedure

Prior to the initial baseline recording period and stress tests, all subjects were given an explanation of the EMG procedures, the muscle sites were prepared by cleaning with an alcohol swab, and the skin was lightly abraded. The resistance between the electrodes was measured with a Radio Shack digital ohmmeter and was kept under 10K ohms. If the resistance was found to be higher, the affected electrodes were removed and the skin preparation was repeated until acceptable resistance levels were obtained with fresh electrodes. The masseter electrodes were placed according to the standard masseter placement described by Lippold (1967). The temporalis electrodes were placed over the anterior portion of the muscle, level with the eyebrow and as close to the hairline as possible. The common reference electrode was placed over the spinous process of the seventh cervical vertebra.

Baseline and Stress Profile

For the baseline recording period and the stress tests, all subjects were instructed to sit in an upright, balanced position with eyes closed and feet flat on the floor in order to minimize movement artifact. The subjects were allowed to open their eyes between data collection periods, but they could not move from the chair.

The first step in the stress profile was a six minute resting baseline. The subjects were instructed to simply relax as deeply as possible during the recording period. No reference was made to the jaws or facial expression as the intent was to avoid artificially influencing habitual orofacial patterns. The experimenter left the room during the baseline recording period.

The first stressor was presented immediately after the resting baseline recording and consisted of a standard two minute mental arithmetic exercise involving the rapid serial subtraction of 7 from 1000. Subjects were told to proceed as quickly as possible, silently, and without making mistakes. This mental task is commonly used in psychophysiologic assessment and is generally considered an effective psychological stressor which results in increased physiological arousal (Lorens & Darrow, 1962).

For the second stressor the subjects were instructed to extend their legs and squeeze them together as hard as possible for a one minute recording period. This stressor was adapted from Kydd (1959) who found that irrelevant masseter and temporalis muscle activity increased dramatically in TMJ subjects, but not in normal controls who exerted maximum force with a foot against a stationary horizontal bar.

The third stressor consisted of eating a cracker and drinking a glass of water followed by three minutes of relaxation. EMG recordings were obtained only during the postmastication relaxation period.

No auditory or visual feedback was provided during any part of the baseline and stress test. The initial appointment including briefing, EMG assessment, and distribution of materials was completed within a standard 50-60 minute clinical appointment. A full account of the baseline and stress profile procedure, recording period lengths, and verbal instructions is offered in Appendix H. Upon completion of the initial EMG assessment session, the subjects were provided with daily symptom charts, the biofeedback training or follow-up appointments were scheduled, and the psychological tests were administered.

Biofeedback Training Protocol

The weekly and daily biofeedback training groups followed identical treatment protocols consisting of 5 one-hour biofeedback training sessions. Four EMGs were routinely used for each of the five sessions, and the muscle/channel assignment was standardized as described above. The initial skin preparation requirements were also maintained throughout. Each of the five biofeedback training sessions consisted of an initial six-minute resting baseline, sitting upright with eyes closed. This was followed by 4 six-minute training periods using the site of highest baseline EMG as the target muscle. The subjects were initially instructed to relax and attempt to reduce the EMG activity of the selected site as much as possible. As training progressed, an emphasis on balancing the EMG activity between sites, where appropriate, was added. Feedback was provided visually by means of the standard Biocomp bar graph display. In this modality, the selected site is displayed as a vertical bar which varies proportionally with the EMG activity. The activity of each of the four muscle sites is also displayed in digital format at the bottom of the screen. The training for balancing between muscles utilized the digital feedback. Thus, it was possible for subjects to receive visual feedback from only the target muscle by focussing on the bar graph, or from all four sites simultaneously by observing the digital display. Auditory feedback was provided with the Biocomp "pure tone" setting. In this modality, the pitch of the tone varies proportionally with the level of EMG activity--rising as the EMG signal increases, and decreasing in pitch as the muscle relaxes. As with the bar graph, only the target muscle received auditory feedback.

The weekly group began biofeedback training after a one-week baseline symptom charting period following the initial appointment. Their training appointments were scheduled to fall within 6-8 days of each other. Symptom charting was continued for the duration of the five-week biofeedback training program. At the conclusion of the first biofeedback training appointment, subjects were given a cassette tape containing the progressive relaxation routine (see Appendix I) and instructed to practice it no less than three times per week for the duration of the treatment program.

The daily biofeedback group received their training along the same protocol as the weekly group except that their sessions were scheduled over five consecutive days. Symptom charting began at the initial baseline session and continued for six weeks. The biofeedback training commenced in the sixth week following the initial appointment with the preceding five weeks of symptom charting serving as a baseline period. The progressive relaxation tape was provided at the conclusion of the first biofeedback training appointment and the subjects were instructed to use it once daily, before or after the biofeedback sessions.

For both biofeedback groups, the initial stressors were repeated following the fourth training period of the fifth biofeedback session. For the relaxation and control groups the baseline EMG and stress profile was repeated at their follow-up sessions 6 and 5 weeks later, respectively. An account of the verbal instructions provided during the biofeedback training sessions is presented in Appendix J.

Relaxation Training Protocol

At the end of the initial EMG baseline appointment, the relaxation group subjects were given a cassette tape containing the same progressive relaxation program used by the biofeedback group. They were instructed to chart their symptoms daily for one week prior to commencing relaxation training, and to continue charting for the six week duration of the study. From weeks 2 through 6 they were to practice the relaxation routine no less than three times per week. As the relaxation subjects were not attending regular office appointments, they were asked to contact the office on a weekly basis to provide an update on their progress and deal with any questions or concerns. The second EMG assessment appointment was scheduled six weeks following the first.

Control Group Protocol

The control group subjects were given symptom charts at the initial EMG assessment appointment and instructed to record their pain symptoms four times daily for five consecutive weeks. As with the relaxation group, they were asked to contact the office on a weekly basis to provide an update on their progress and an opportunity to discuss any concerns. The control group received no biofeedback or relaxation training during the five week symptom charting period, however, they were offered the option of completing their choice of the biofeedback or relaxation training protocols upon completion of the study requirements.

After the subjects had received the posttreatment clinical examination, they attended a debriefing appointment during which the results of the MMPI and MBTI were discussed. Assuming all aspects of the treatment program had been satisfactorily completed, each subject had her deposit refunded. At the conclusion of the debriefing the biofeedback and relaxation group subjects completed the posttreatment questionnaire which they left with the receptionist. The posttreatment questionnaire is reproduced in Appendix K.

IV. RESULTS

The analysis of data will be presented under three different groupings:

A. Descriptive statistics:

i. Subject characteristics as determined from the screening questionnaire.

ii. Occlusal, joint, and muscle characteristics from the clinical examinations.

iii. EMG data collected during the baseline and stressors.

iv. Personality characteristics.

B. Treatment outcome:

i. EMG pre and posttreatment.

ii. Pre and posttreatment joint and muscle palpation data.

iii. Baseline and treatment phase pain symptom charts.

iv. Posttreatment questionnaire responses.

C. Correlations:

i. EMG data

  a. EMG activity and palpation pain scores.
  b. Symptom chart rating and EMG activity.
  c. EMG activity and subjective improvement rating.
  d. EMG and MMPI scales.

ii. Clinical examination

  a. Pretreatment cumulative palpation pain and specific palpation sites.

  - b. Occlusal characteristics.
  - c. Palpation pain and daily self-report pain.
  - d. Palpation pain rating and MMPI.

iii. Screening questionnaire and percentage improvement.

iv. Posttreatment questionnaire.

iv. MMPI

  - a. MMPI and weekly pain symptom rating.
  - b. MMPI and percentage improvement.

v. MBTI frequency distribution.

A. Descriptive Statistics

Subject Characteristics

The characteristics of the subject population as reported through the screening questionnaire are presented in Table 1. As compared to the responses obtained in Gervais (1984) using the same questionnaire, the present patient population was older and substantially more symptomatic in virtually all respects. The presence of clenching or bruxing was noted in over 70% of TMJ patients as compared to 45-54% of the subjects in the earlier study. Similarly, the symptom of chronic headaches, neck, and shoulder pain was a major complaint in 86% of the patient sample as compared to 54% in the previous study. Symptoms directly implicating the temporomandibular joint, i.e. clicking, limited opening, and pain, were reported in over 90% of the participants as contrasted with 75-79%

previously. The presence of tinnitus was also increased to 36% from 16%. Overall, the results of the screening questionnaire indicate that the patients included in this study reported significantly more signs and symptoms of TMJ pain and dysfunction than the subjects assessed in Gervais (1984). An account of the questionnaire responses is provided in Table 1.

Table 1

Screening Questionnaire

Characteristics of study population (N=100)

	Mean Age	Range	S.D.
	35.0	15-79	11.8

	% Yes
1. Do you clench or grind your teeth during the day?	70.7
2. Have you been made aware of clenching or grinding your teeth during the night?	71.4
3. Do you have chronic headaches, or neck and shoulder pains?	86.5
4. Do you frequently have gastrointestinal disturbances?	48.4
5. Do you ever wake up with an awareness of, or about, your teeth or jaw like you had them clenched in your sleep?	72.7
6. Do you have any awareness of the muscles of your neck or shoulders?	96.9
7. Do you have a tight or stiff neck?	93.7
8. Do you now, or have you ever had, pain in your jaw joint or the sides of your face (in and about the ears)?	91.9

9. Do you have a clicking jaw joint or have you ever experienced an inability to move your jaw or open your mouth widely?		92.0
10. Which side of your mouth do you chew on?	Both	62.5
11. Do you tend to breathe mostly through your nose?		76.0
12. Are you aware of persistent ringing in your ears?		36.7
18. Is your condition the result of an injury or accident?		38.8
19. Are you presently involved in legal action regarding your condition?		15.2
20. How long have you had jaw problems? (years)	Mean: 7.95 Range: 1-50 S.D.: 9.34	

Clinical Examination

Clinical examinations, pre and posttreatment, were obtained for nearly all patients in the study. The findings will be presented in three categories:

a. occlusal characteristics;

b. joint sounds;

c. palpation scores.

Occlusal Characteristics

Over half of the patients (56-64%) displayed Class I occlusion according to the Angle classification system. Approximately 25-30% fell into the Class II category, and approximately 10% possessed Class III occlusion. Overbite ranged from 0.0-8.0 mm with a mean of

2.84mm. The range of maximum opening was 19.0-55.0mm with a mean of 38.33mm. Pain upon maximum opening was reported in 26% of the sample. A more detailed analysis of the occlusal data is presented in Table 2.

Table 2

Occlusal Data (Pretreatment)

	Angle Classification (valid % of sample N=81) I	II	III
Molar (R)	56.3	33.7	8.7
Molar (L)	63.0	24.7	11.1
Canine (R)	58.0	30.9	11.1
Canine (L)	64.2	25.9	9.9

Other characteristics

	Range	Mean	S.D.
Overbite:	0.0-8.0mm	2.84mm	1.89mm
Maximum opening:	19.0-55.0mm	38.33mm	7.50mm
Painful to open to maximum: (% Yes)		26.0	

Joint Sounds

The clinical examination registered sounds for each TMJ during the opening and closing phases of the mandibular movement cycle. A four-point rating scale was used to assess the severity of the sounds: 0=absence of noise, 1=mild, 2=moderate, 3=severe. No formal procedures were used to control inter-rater reliability, however, clinicians were to base their assessment of joint noise or pain on their professional judgement and expertise in diagnosing and treating TMJ disorders. No radiographs of other diagnostic procedures were carried out to further evaluate the nature of the observed joint sounds. A summary of joints sounds is presented in Table 3.

Table 3

Pretreatment Joint Sounds

Location and type of sound	Severity	Valid percent of sample (N=87)	
		Open	Close
Popping L	0	66.7	72.4
	1	13.8	11.5
	2	12.6	11.5
	3	6.9	4.6
Popping R	0	67.8	74.7
	1	11.5	10.3
	2	14.9	11.5
	3	5.7	3.4
Soft tissue grating L	0	77.0	80.5
	1	16.1	11.5
	2	5.7	6.9
	3	1.1	1.1
Soft tissue grating R	0	79.3	82.8
	1	12.6	11.5
	2	6.9	4.6
	3	1.1	1.1
Hard tissue grating L	0	72.4	77.0
	1	10.3	6.9
	2	8.0	8.0
	3	9.2	8.0
Hard tissue grating R	0	78.2	80.5
	1	8.0	5.7
	2	5.7	6.9
	3	8.0	6.9

In order to facilitate the analysis of pre and posttreatment effects, each patient's joint noise scores were also summed to provide a cumulative noise rating index. This noise index is reproduced in Table 4.

Table 4

Pretreatment Noise Scores

Cumulative Score	Valid Percent of Sample (N=100)
0	34.0
1-5	33.0
6-10	24.0
11-36	9.0

Mean: 4.43 S.D.: 5.59

Palpation Scores

Temporomandibular Joint

In the clinical examination the temporomandibular joints were palpated both laterally and posteriorly, with and without movement. The same four-point rating scale was used to assess pain or tenderness. Approximately 80% of patients reported some degree of pain to lateral or posterior palpation of the temporomandibular joints. Detailed palpation results are documented in Table 5.

Table 5

Pretreatment Palpation Scores TM-Joint (N=87)

	Without Movement Severity	% of sample	With Movement Severity	% of sample
TMJ lateral (L)	0	63.2	0	72.4
	1	23.0	1	16.1
	2	9.2	2	5.7
	3	4.6	3	5.7
TMJ lateral (R)	0	69.0	0	66.7
	1	13.8	1	11.5
	2	13.8	2	13.8
	3	3.4	3	8.0
TMJ posterior (L)	0	69.0	0	74.7
	1	13.8	1	16.1
	2	13.8	2	6.9
	3	3.4	3	2.3
TMJ posterior (R)	0	66.7	0	70.1
	1	13.8	1	17.2
	2	16.1	2	10.3
	3	3.4	3	2.3

Muscles

Muscle tenderness was also evaluated on the four-point scale. The muscles were individually palpated and assigned scores which are reported in detail in Table 6. Overall, the most frequently painful muscle was the left lateral pterygoid which was tender in 59.8% of the patient sample. This was followed by the left deep masseter (57.5%), the right deep masseter (56.3%), and the left medial pterygoid (55.2%). A number of other muscle sites were also tender in over 50% of the patients. Sharav, Tzukert, and Rafaeli (1978) report similar incidences of muscle tenderness.

As with the noise scores, the TMJ and muscle palpation scores were also summed to produce a cumulative palpation pain index for the group. The cumulative palpation score for the group ranged from 0.0 to 84, rendering a mean of 18.24 with a standard deviation of 17.76.

Table 6

Pretreatment Muscle Palpation Scores (N=87)

Muscle %	Severity	Valid % of sample	Cumulative (1-3)
Post. temporalis (L)	0	63.2	
	1	18.4	
	2	11.5	
	3	6.9	36.8
Post. temporalis (R)	0	63.2	
	1	16.1	
	2	14.9	
	3	5.7	36.8
Ant. temporalis (L)	0	57.5	
	1	20.7	
	2	13.8	
	3	8.0	42.5
Ant. temporalis (R)	0	55.2	
	1	18.4	
	2	19.5	
	3	6.9	44.8
Superficial masseter (L)	0	48.3	
	1	17.2	
	2	24.1	
	3	10.3	51.7

Superficial masseter (R)	0	46.0	
	1	17.2	
	2	27.6	
	3	9.2	54.0
Deep masseter (L)	0	42.5	
	1	21.8	
	2	25.3	
	3	10.3	57.5
Deep masseter (R)	0	43.7	
	1	21.8	
	2	25.3	
	3	9.2	56.3
Hyoid, ant. digastric (L)	0	72.4	
	1	13.8	
	2	8.0	
	3	5.7	27.5
Hyoid, ant. digastric (R)	0	73.6	
	1	13.8	
	2	6.9	
	3	5.7	26.4
Sternomastoid (L)	0	48.3	
	1	17.2	
	2	23.0	
	3	11.5	51.7
Sternomastoid (R)	0	49.4	
	1	18.4	
	2	24.1	
	3	8.0	50.6
Suboccipital area (L)	0	46.0	
	1	20.7	
	2	21.8	
	3	11.5	54.0
Suboccipital area (R)	0	46.0	
	1	23.0	
	2	23.0	
	3	8.0	54.0
Medial pterygoid (L)	0	44.8	
	1	26.4	
	2	19.5	
	3	9.2	55.2

Medial pterygoid (R)	0	48.3	
	1	24.1	
	2	19.5	
	3	8.0	51.7
Lateral pterygoid (L)	0	40.2	
	1	25.3	
	2	14.9	
	3	19.5	59.8
Lateral pterygoid (R)	0	47.1	
	1	21.8	
	2	14.9	
	3	16.1	52.9
Temporal tendon (L)	0	58.6	
	1	17.2	
	2	12.6	
	3	11.5	41.4
Temporal tendon (R)	0	60.9	
	1	13.8	
	2	17.2	
	3	8.0	39.1

EMG Data Collected During Baseline and Stressors

Temporalis and masseter EMG activity was recorded simultaneously from each muscle throughout all phases of the baseline and stress tests, as well as during biofeedback training. Each muscle occupied a specific channel in the recording apparatus. The accompanying software program calculated the maximum, minimum, mean, and standard deviation for each muscle over the recording period. For the purposes of this discussion, only the mean EMG activity from each muscle was used in the statistical analysis. The observed baseline EMG activity in the temporalis and masseter muscles is consistent with that reported in Gervais, Fitzsimmons, and Thomas, (1989).

Analyses of variance between the baseline EMG means and those recorded during the stressors were significant (p<.0001), indicating that the presentation of the stressors resulted in a significant variation from baseline in all muscles. There was also a significant difference (p<.0001) between the muscle types, i.e. temporalis and masseter, but not between the left or right temporalis or masseter (p>.05). The EMG means for the baseline and stress conditions are reported in Table 7.

Table 7

Channel Means for each Experimental Condition
(EMG microvolts) N=100

	L. temp.	L. mass.	R. temp.	R. mass.
Baseline				
Mean:	5.671	4.579	5.309	3.656
S.D.:	4.355	3.328	3.359	2.410
Serial subtraction				
Mean:	6.375	4.599	5.973	3.629
S.D.:	4.639	3.133	3.464	2.356
Physical exertion				
Mean:	7.351	5.683	6.360	4.410
S.D.	6.498	5.026	4.214	3.917
Post mastication				
Mean:	4.964	4.028	4.777	3.477
S.D.	3.920	2.812	2.965	2.404

F(1.7, 159.4) = 25.77, p<.0001

Personality Characteristics

MMPI

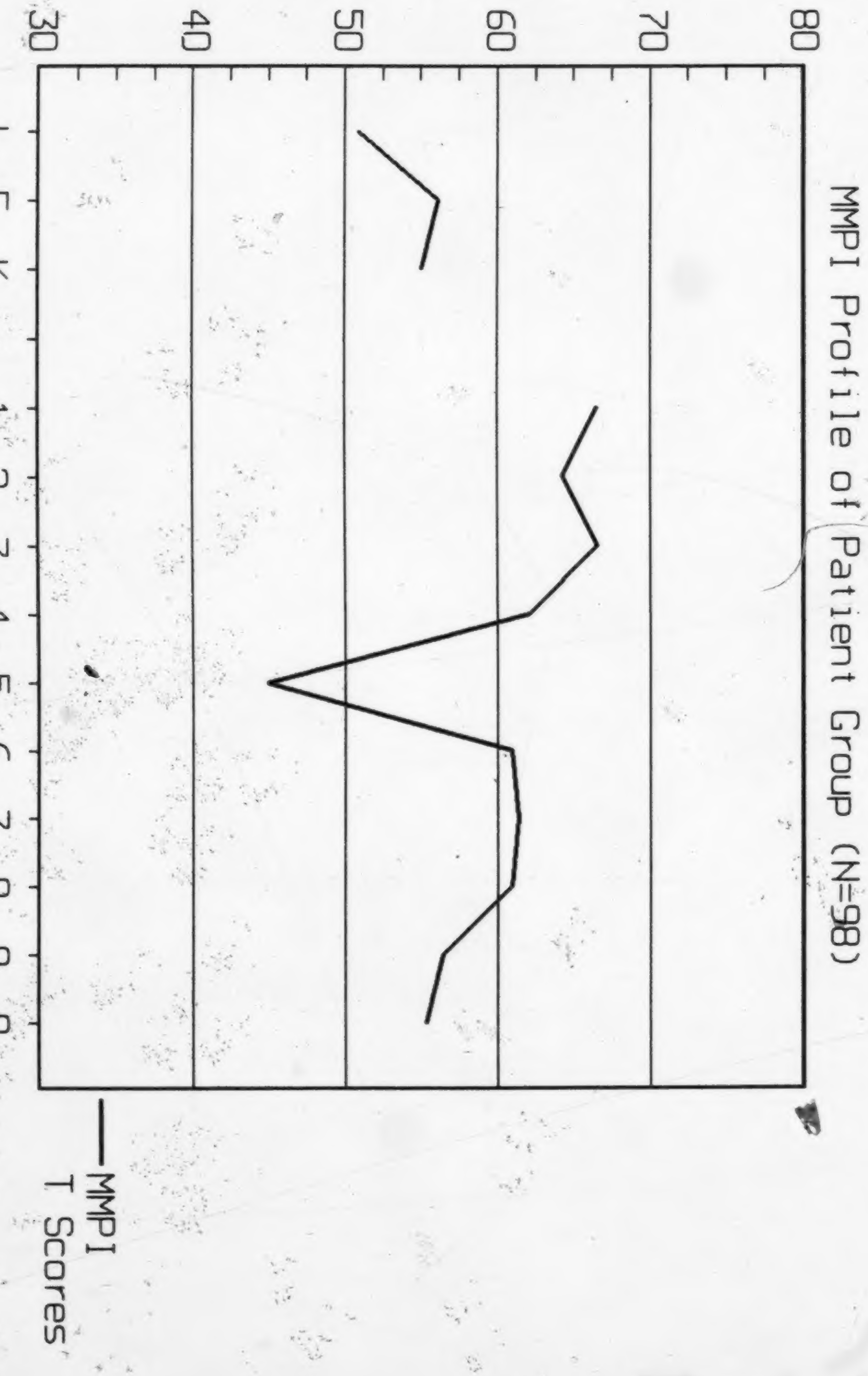
A preliminary analysis of the MMPI T scores revealed validity scale scores in the normal range and mild to borderline elevations on seven of the standard clinical scales. Most prominent were scales 3(Hysteria) (T=66.57), 1 (Hypochondriasis) (T=66.51), and 2 (Depression) (T=64.29). This type of profile has frequently been associated with the chronic pain patient (Golden, 1979; Merskey, 1986), and has been reported in TMJ patients by a number of investigators (Schwartz, Greene, & Laskin, 1974; Alpern, Nuelle, & Wharton, 1988; Schumann, Zwiener, & Nebrich, 1988). Scale 4 (Psychopathic Deviate) also registered a borderline elevation (T=62.07) as did scale 7 (Psychasthenia) (T=61.40). The MMPI T scores for the patient group are reported in Table 8 and graphed in Figure 3.

Table 8

MMPI T Scores for Patient Group
(N=98)

Scale	Mean	S.D.
L	50.87	6.55
F	56.11	8.10
K	55.02	8.54
1 (Hs)	66.51	12.42
2 (D)	64.29	11.84
3 (Hy)	66.57	11.71
4 (Pd)	62.07	12.41
5 (Mf)	44.88	9.42
6 (Pa)	60.93	9.91
7 (Pt)	61.40	10.29
8 (Sc)	60.96	11.77
9 (Ma)	56.42	12.16
0 (Si)	55.29	11.04

Figure 3


MMPI Profile of Patient Group (N=98)
80
70
60
50
40
30
L
F
K
1
2
3
4
5
6
7
8
9
0
MMPI T Scores

The MMPI data were subsequently sorted according to the P-A-I-N typology criteria proposed by Costello, Hulsey, Schoenfeld, and Ramamurthy (1987) in order to ascertain whether the distribution of the patients in the present study would conform to the estimated base rates the authors propose for the four pain types. According to their classification rules, only profile type N, "normal" (26%) corresponded to the predicted base rate of 25%. Profile types P (6%), A (4%), and I (3%) were markedly under-represented in this sample when compared to the estimated base rates of 15%, 20%, and 30%, respectively.

The MMPI data were further sorted manually for the presence of a 'conversion V'(type A) profile type, i.e. scales 1 and 3 > scale 2. Although the 'V' may not have been as pronounced as that defined by Costello et al. (Hs + Hy > 2D by 15 T or more), approximately 23% of the patient profiles fell into this category. This frequency is more consistent with the estimated base rate and suggests that, for practical purposes, the Costello et al. classification criteria may be excessively stringent and could be applied with more flexibility. Deiter, and Swerdlow (1988), for example, used the following formula in their definition of a conversion 'V': 1(Hs) = 65 T or more and > than 2(D) by 5 T or more;

and 3(Hy) > 2(D) by at least 10 T. Similarly, manual sorts selected 14% of the group for type P, 7% for type I, and 36% for type N. Roughly 14% of the profiles were unclassifiable according to the P-A-I-N typology, and 4% were in the normal range with the exception of a spike 2(D).

MBTI

The Myers-Briggs Type Indicator was completed by 94 of the subjects. The most frequently observed types were ISFJ (N=15), ENFP (N=15), ISTJ (N=10), and ESFJ (N=13). No representation was found for the type INTP, and three types were observed only once: ISTP, INTJ, and ENTJ. The distribution of MBTI types produced by the patients in the treatment study is presented in Table 9.

Table 9

MBTI Type Table of TMJ Patients
N=94

ISTJ N=10 10.63%	ISFJ N=15 15.95%	INFJ N=6 6.38%	INTJ N=1 1.06%
ISTP N=1 1.06%	ISFP N=4 4.25%	INFP N=9 9.57%	INTP N=0 0.00%
ESTP N=4 4.25%	ESFP N=3 3.19%	ENFP N=15 15.95%	ENTP N=5 5.31%
ESTJ N=4 4.25%	ESFJ N=13 13.82%	ENFJ N=3 3.19%	ENTJ N=1 1.06%

B. Treatment Outcome

EMG Pre and Posttreatment

The 100 patients in the study were sequentially assigned to the three treatment groups or control group as described above in Chapter 3. Pretreatment EMG recordings from the temporalis and masseter muscles were obtained for each subject during baseline and three stressor conditions. Posttreatment EMG recordings were obtained using the same procedure at the end of the fifth biofeedback training session for the weekly and daily biofeedback groups, and at the second office visit following the five week relaxation group program and after the five week control group period. The pre and posttreatment mean EMG values for each muscle are reported in Tables 10.1-10.4.

Analyses of variance between pre and posttreatment baseline and stressor conditions revealed that all groups displayed a significant reduction in masseter and temporalis EMG activity over time ($p<.05$). The stress conditions were also associated with a significant change in EMG activity in all muscles, pre and posttreatment ($p<.0001$). Hence, no significant change in stress response, as determined by variation of EMG activity from baseline, was noted following the treatment or control period ($p>.05$). There was, however, a near significant change in the differential response to the stressors between the masseter and temporalis (p=.06). No significant difference was found in the stress response from the left or right sides (p>.05).

Table 10.1

Pre and Posttreatment EMG Microvolts
Weekly Biofeedback (N=26)

	Pre		Post	
	Mean	S.D.	Mean	S.D.
Baseline				
L. temporalis	5.360	2.977	3.604	1.870
L. masseter	4.238	2.575	2.667	1.126
R. temporalis	5.742	2.853	3.137	1.718
R. masseter	3.790	2.178	2.379	1.243
Serial Subtraction				
L. temporalis	6.121	3.417	4.201	1.984
L. masseter	4.076	2.222	3.140	1.521
R. temporalis	5.781	2.518	4.228	3.152
R. masseter	3.625	2.142	2.573	1.515
Physical Exertion				
L. temporalis	7.524	4.678	5.325	3.050
L. masseter	5.959	4.821	3.741	2.123
R. temporalis	6.923	3.186	4.705	2.901
R. masseter	4.790	3.530	3.090	1.928
Post Mastication				
L. temporalis	5.000	3.036	4.038	2.355
L. masseter	3.868	2.385	3.155	1.750
R. temporalis	5.319	2.625	3.875	2.738
R. masseter	3.780	2.806	2.679	1.909

Table 10.2

Pre and Posttreatment EMG Microvolts
Daily Biofeedback (N=25)

	Pre		Post	
	Mean	S.D.	Mean	S.D.
Baseline				
L. temporalis	5.826	6.512	3.569	3.526
L. masseter	4.708	4.338	2.170	1.493
R. temporalis	4.695	4.441	3.861	5.521
R. masseter	3.921	3.225	1.931	1.242
Serial Subtraction				
L. temporalis	7.345	7.135	4.172	3.176
L. masseter	5.444	4.751	2.975	1.647
R. temporalis	6.945	5.541	4.567	5.930
R. masseter	4.148	3.431	2.196	1.044
Physical Exertion				
L. temporalis	7.175	7.902	4.584	3.574
L. masseter	5.080	5.322	3.026	1.704
R. temporalis	5.692	5.415	4.801	6.616
R. masseter	4.288	4.213	2.327	1.017
Post Mastication				
L. temporalis	5.382	5.940	3.611	3.623
L. masseter	4.548	3.975	2.513	1.936
R. temporalis	4.455	4.050	4.117	6.368
R. masseter	3.896	3.173	2.026	1.107

Table 10.3

Pre and Posttreatment EMG Microvolts
Relaxation (N=24)

	Pre		Post	
	Mean	S.D.	Mean	S.D.
Baseline				
L. temporalis	5.954	4.275	6.238	3.985
L. masseter	5.559	3.778	4.806	3.754
R. temporalis	6.109	3.520	4.874	3.232
R. masseter	3.599	1.845	3.027	1.650
Serial Subtraction				
L. temporalis	6.140	3.846	6.290	3.416
L. masseter	4.939	2.930	4.409	2.927
R. temporalis	5.882	2.541	5.443	3.608
R. masseter	3.367	1.261	3.013	1.586
Physical Exertion				
L. temporalis	7.111	5.487	8.473	7.754
L. masseter	7.041	6.192	6.055	5.118
R. temporalis	7.139	4.699	6.054	4.440
R. masseter	4.512	3.720	3.602	1.820
Post Mastication				
L. temporalis	4.912	3.474	5.727	3.675
L. masseter	4.506	2.821	4.227	3.600
R. temporalis	4.957	2.906	4.392	2.969
R. masseter	3.245	1.500	2.850	1.663

Table 10.4

Pre and Posttreatment EMG Microvolts
Control (N=25)

	Pre		Post	
	Mean	S.D.	Mean	S.D.
Baseline				
L. temporalis	5.574	3.141	3.651	2.412
L. masseter	3.871	2.232	4.006	2.550
R. temporalis	4.680	2.279	3.097	1.679
R. masseter	3.317	2.309	2.371	1.001
Serial Subtraction				
L. temporalis	5.933	3.391	4.204	2.728
L. masseter	4.005	1.896	4.190	2.503
R. temporalis	5.387	2.326	3.872	2.494
R. masseter	3.386	2.165	2.585	1.188
Physical Exertion				
L. temporalis	7.573	7.794	5.175	3.816
L. masseter	4.671	3.417	4.364	2.369
R. temporalis	5.667	3.283	4.284	3.289
R. masseter	4.034	4.369	2.812	1.376
Post Mastication				
L. temporalis	4.574	2.726	3.313	2.055
L. masseter	3.236	1.553	3.847	2.711
R. temporalis	4.352	2.063	2.949	1.601
R. masseter	2.984	1.736	2.435	0.950

Pre and Posttreatment Joint and Muscle Palpation

As was indicated above, the muscle and TMJ palpation pain and noise scores were summed to produce cumulative scores in order to facilitate the analysis and presentation of results. Using multivariate analysis, no significant differences between the groups were found in cumulative noise (p>.05) and pain scores (p>.05) at the pre and posttreatment examination. This was verified by a subsequent analysis of covariance. However, all groups demonstrated a significant reduction in palpation pain ($p<.001$) and TMJ noise scores ($p<.05$) over time. While an improvement in clinically assessed TMJ dysfunction signs and symptoms was found in all groups, the greatest degree of change was noted in the weekly biofeedback group with a 62.14% reduction in palpation pain. This was followed by the relaxation group with a 34.68% decrease. The daily biofeedback group registered the least amount of improvement with a pain reduction of 18.31%. A 34.39% decrease in pain symptoms was found in the control group. No change was observed in noise scores for the daily biofeedback group. Conversely, a 50.0% reduction in noise was found in the relaxation group. The weekly biofeedback and control groups demonstrated similar reductions in joint noise of 34.25% and 36.73%, respectively. As was indicated above, however, these differences in percentage change were not statistically significant. A complete account of the pre and posttreatment cumulative noise and palpation pain scores is presented in Table 11, and Figures 4 and 5.

Table 11

Pre and Post Cumulative TMJ Noise and Muscle Pain Scores

	Noise		Pain	
	Pre	Post	Pre	Post
Total Group	4.430	3.010	18.240	11.300
Weekly Biofeedback	4.154	2.731	20.115	7.615
Daily Biofeedback	3.960	3.960	19.880	16.240
Relaxation	5.750	2.875	21.625	14.125
Control	3.920	2.480	11.400	7.480

Noise: F(3,96) = 1.12; p>.05 (Pre N=87)
Pain: F(3,96) = 1.57; p>.05 (Post N=84)

Figure 4

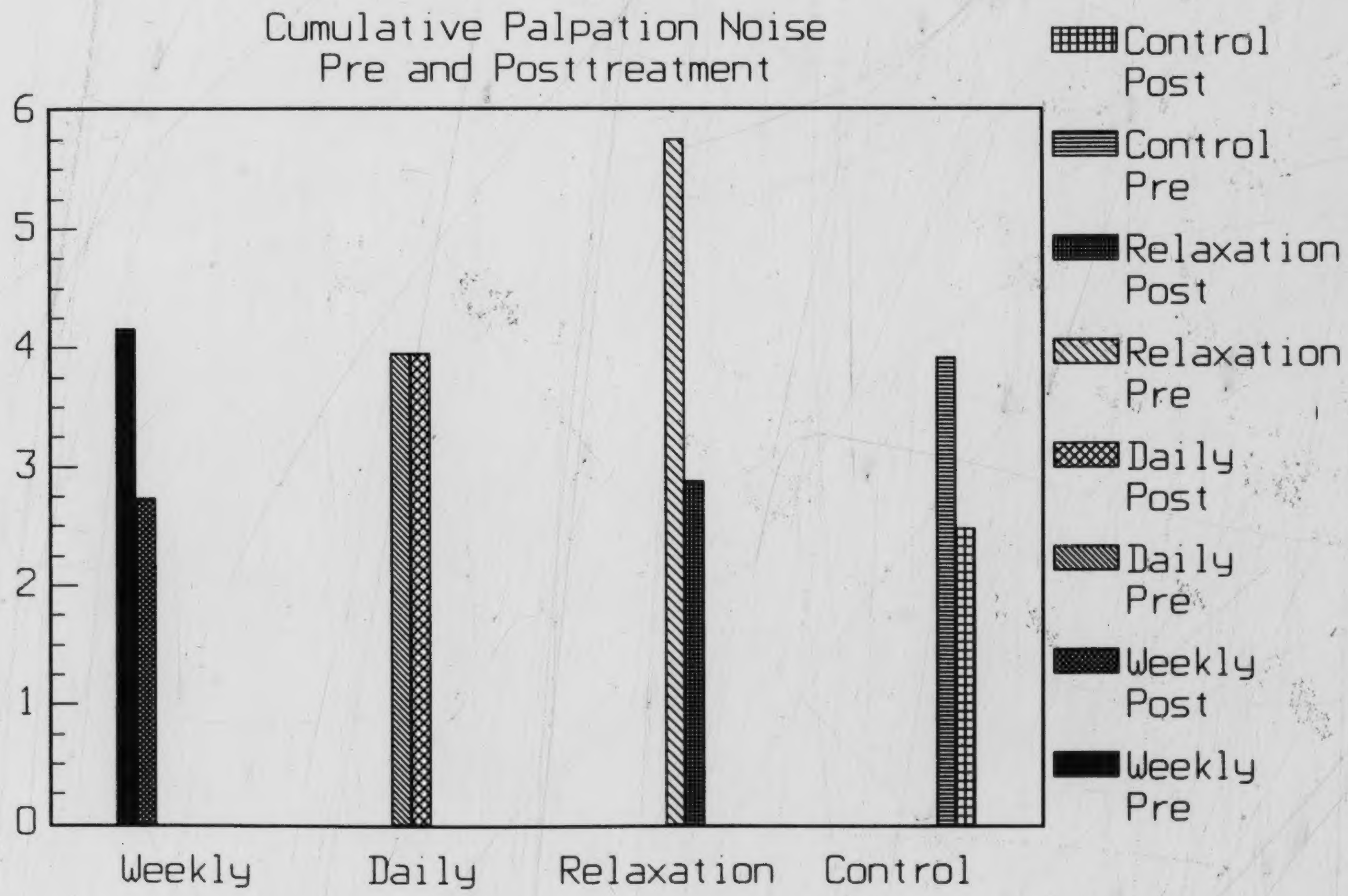
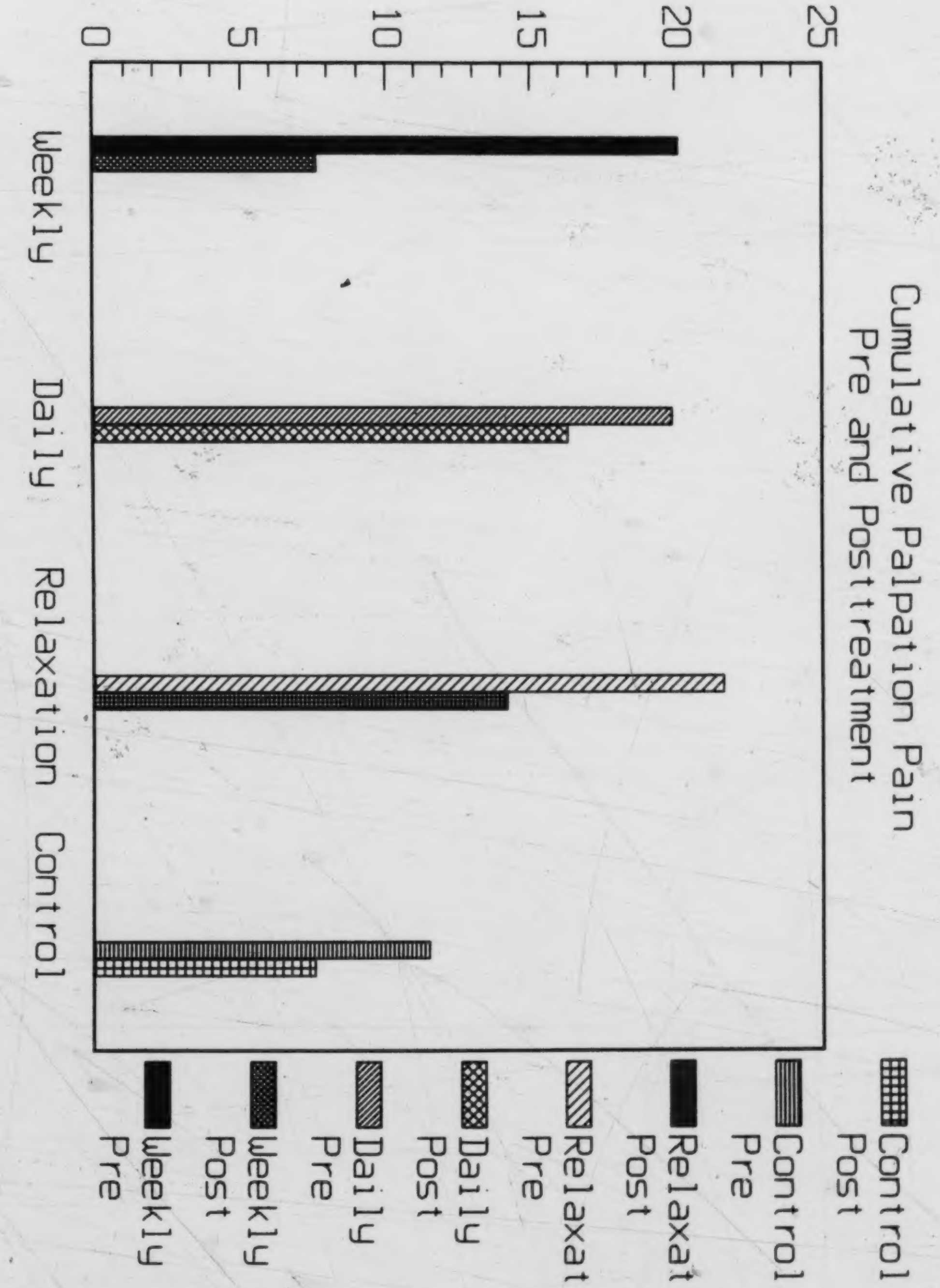

Cumulative Palpation Noise
Pre and Posttreatment
6
5
4
3
2
1
0
Weekly
Daily
Relaxation
Control
Control Post
Control Pre
Relaxation Post
Relaxation Pre
Daily Post
Daily Pre
Weekly Post
Weekly Pre

Figure 5


Cumulative Palpation Pain
Pre and Posttreatment
0
5
10
15
20
25
Weekly
Daily
Relaxation
Control
Control Post
Control Pre
Relaxation Post
Relaxation Pre
Daily Post
Daily Pre
Weekly Post
Weekly Pre

Daily Pain Symptom Chart

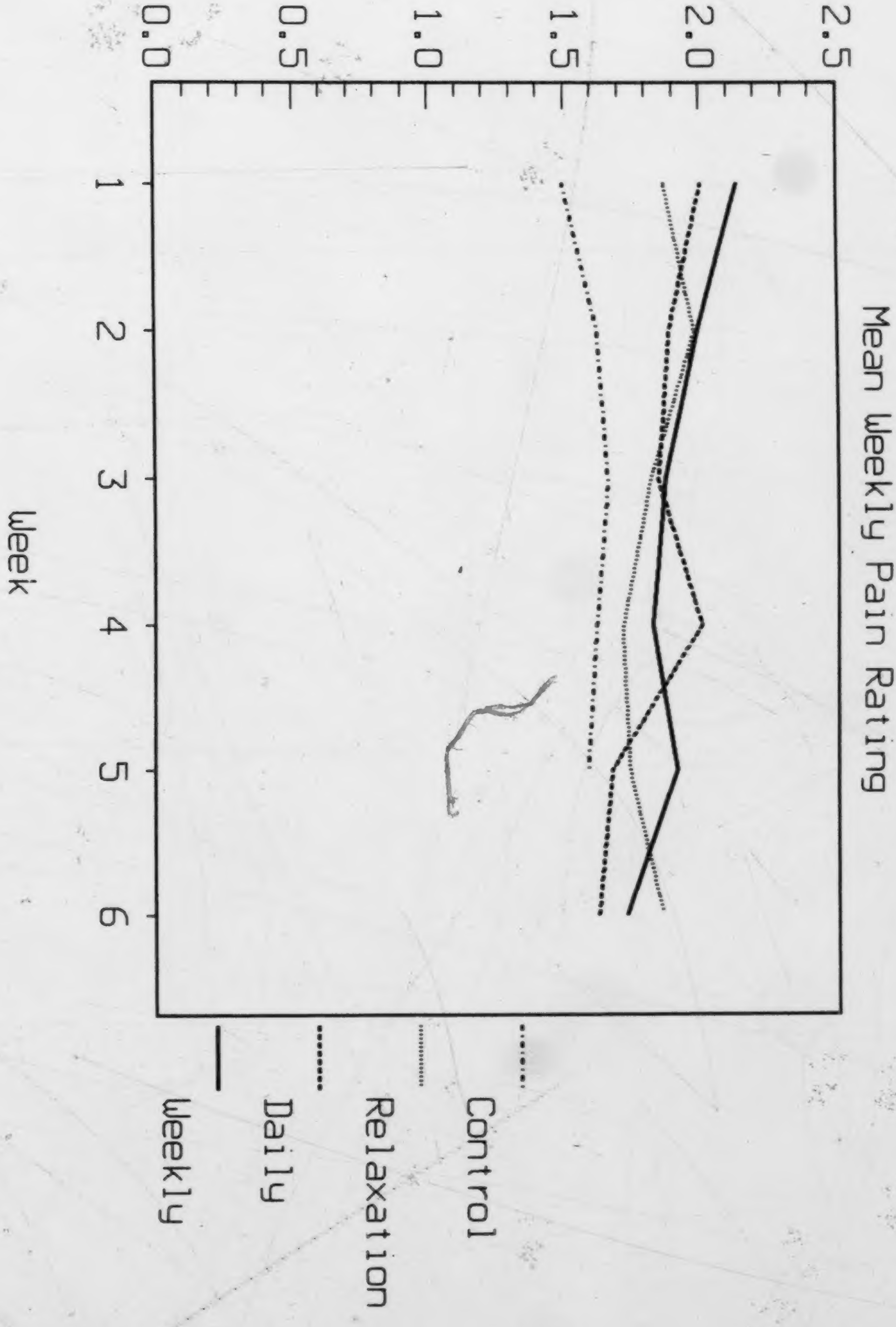
An analysis of the daily pain charts kept by all participants revealed no significant differences between the treatment and control groups (p>.05), nor from week to week (p>.05). A summary of the mean weekly chart scores is presented in Table 12 and Figure 6.

Table 12

Mean Weekly Pain Rating

	Weekly (N=26)	Daily (N=25)	Relax (N=24)	Control (N=25)
Week 1	2.136	2.003	1.872	1.498
Week 2	1.988	1.881	1.978	1.617
Week 3	1.866	1.837	1.811	1.656
Week 4	1.819	2.001	1.709	1.611
Week 5	1.909	1.669	1.738	1.580
Week 6	1.724	1.619	1.857	N/A

Figure 6


Mean Weekly Pain Rating
2.5
2.0
1.5
1.0
0.5
0.0
1
2
3
4
5
6
Week
Control
Relaxation
Daily
Weekly

Posttreatment Questionnaire Responses

A posttreatment questionnaire was completed by the biofeedback and relaxation group subjects at the debriefing session held one to three weeks after the conclusion of the treatment program, and following the posttreatment clinical examination by the referring dentist. The intent of the questionnaire was to obtain the subjects' personal evaluation of the treatment they had received, i.e., whether their participation in the treatment program had made a significant difference in their condition. This subjective assessment was expressed in terms of percentage improvement or worsening of symptoms, and was one of the principle measures of treatment outcome in addition to the pre and posttreatment EMG recordings, clinical examinations, and the daily pain symptom chart.

Overall, 51 of the 74 treatment condition subjects (weekly, daily, relaxation), or 68.91%, reported a subjective improvement in their TMJ-related symptoms following treatment program. In 20 of the treatment condition participants (27.02%), no subjective change was reported, and 3 rated their condition as worse following treatment. There was a significant difference in percentage improvement between the groups ($p<.05$), with the weekly biofeedback group reporting a mean symptom improvement of 45.38% in contrast to the daily biofeedback condition at 26.20%, and the relaxation group at 27.38%. A complete account of the subjective degree of improvement following treatment for the biofeedback and relaxation groups is contained in Table 13 and Figure 7.

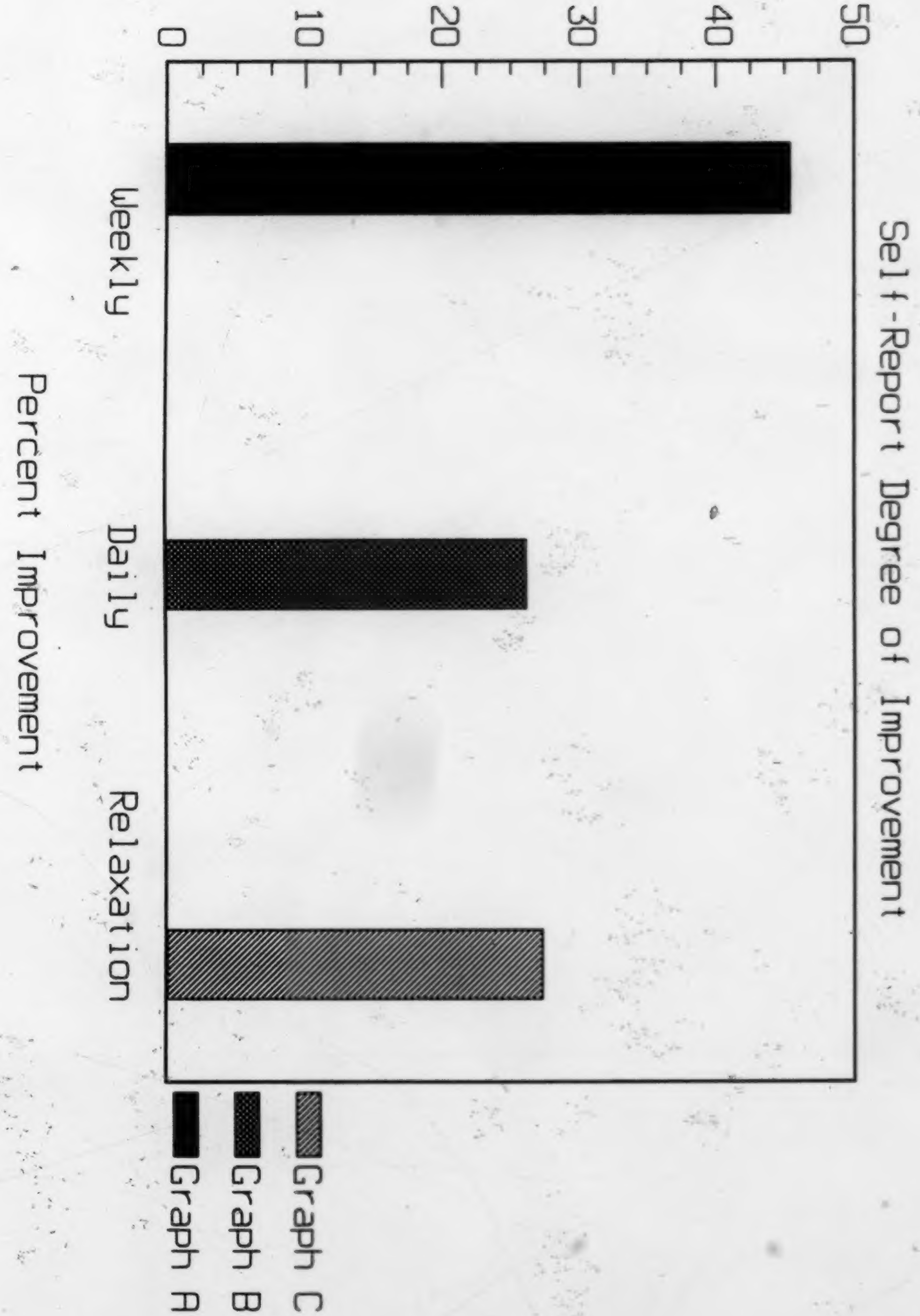
Table 13

Subjective Improvement Rating

Group	Mean %	S.D.
Weekly Biofeedback	45.38	32.73
Daily Biofeedback	26.20	24.20
Relaxation	27.38	26.53

F(3,56)=3.67, p<.05

Figure 7


Self-Report Degree of Improvement
0
10
20
30
40
50
Weekly
Daily
Relaxation
Percent Improvement
Graph C
Graph B
Graph A

There was no significant difference in frequency of home relaxation practice between the treatment groups (p>.05). The weekly biofeedback and relaxation participants were instructed to use the relaxation tape three times per week and reported mean frequencies of practice of 3.46 (S.D.=1.24) and 3.72 (S.D.=1.66) times per week. The daily biofeedback participants were to practice the relaxation exercises on a daily basis and reported a mean frequency of 4.74 (S.D.=1.79) times per week. On the basis of self-report, the patients in the study appear to have been reasonably compliant with the home practice requirements. Of the 74 treatment subjects, 69% indicated that they had benefited from their participation in the study, and 92% intended to continue practicing their relaxation skills on a regular basis.

C. Correlations

EMG Data

In order to simplify the presentation of the EMG correlations, the mean right and left temporalis EMGs were combined to provide pre and posttreatment pooled temporalis EMG means for each of the baseline and stressors. The same procedure was conducted with the masseter EMG data. These pooled means for pre and posttreatment temporalis and masseter EMG were subsequently correlated with the other variables obtained pre and posttreatment.

EMG Activity and Palpation Pain Scores

Significant, moderately strong positive correlations were noted between pre and posttreatment mean baseline EMG activity and the pre and posttreatment cumulative muscle palpation scores for the daily biofeedback group. The strongest correlation was between pretreatment masseter EMG and posttreatment pain (r=.7262). Baseline temporalis and masseter EMG at pretreatment demonstrated stronger correlations with pretreatment pain (r=.7047, r=.5990) than did posttreatment temporalis and masseter EMG correlated with posttreatment pain (r=.5938, r=.3790).

For the remaining three groups, the correlations between pre and posttreatment baseline EMG activity and palpation pain were largely non-significant. The weekly biofeedback group obtained a significant negative correlation between pretreatment baseline masseter EMG and pretreatment pain (r=-.3885), and for the relaxation group, a significant correlation was noted between posttreatment masseter EMG and posttreatment pain (r=.4209). Details of the correlations between pre and posttreatment baseline EMG and palpation pain are presented in Tables 14.1-14.4.

Table 14.1

Mean Baseline EMG and Muscle Palpation Pain
Weekly Biofeedback (N=23/19)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	.1393	.263	-.0801	.372
Masseter (pre)	-.3885	.033	-.3725	.058
Temporalis (post)	.1180	.296	-.0301	.451
Masseter (post)	.0513	.408	.0228	.463

Table 14.2

Mean Baseline EMG and Muscle Palpation Pain
Daily Biofeedback (N=21/23)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	.7047	.000	.7033	.000
Masseter (pre)	.5990	.003	.7262	.000
Temporalis (post)	.6770	.000	.5938	.001
Masseter (post)	.4850	.013	.3790	.037

Table 14.3

Mean Baseline EMG and Muscle Palpation Pain
Relaxation (N=21/20)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	-.1605	.243	.3156	.088
Masseter (pre)	-.0672	.386	.2001	.199
Temporalis (post)	.0016	.497	.1756	.229
Masseter (post)	.3367	.068	.4209	.032

Table 14.4

Mean Baseline EMG and Muscle Palpation Pain
Control (N=22/22)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	.1711	.223	.0308	.446
Masseter (pre)	-.0466	.418	-.2091	.175
Temporalis (post)	.0440	.423	-.2962	.090
Masseter (post)	.1193	.298	-.3511	.055

As was the case with the baseline EMG correlations discussed above, the daily biofeedback group produced significant positive correlations on all pre and posttreatment EMG and pain variables. The strongest of these was between the pretreatment masseter EMG and posttreatment pain (r=.7057). Moderate correlations were noted for both pre and posttreatment EMG and pain.

Generally non-significant correlations were noted between pre and posttreatment pain and EMG. Posttreatment serial subtraction masseter EMG and pain for the relaxation group produced the only other significant correlation. The correlations between pre and posttreatment palpation pain and the EMG activity recorded during the serial subtraction stressor are presented in Tables 15.1-15.4.

Table 15.1

Mean Serial Subtraction EMG
and Muscle Palpation Pain
Weekly Biofeedback (N=23/19)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	.0557	.400	.0142	.477
Masseter (pre)	-.4632	.013	-.3633	.063
Temporalis (post)	.2380	.137	-.0312	.449
Masseter (post)	.3407	.056	.0019	.497

Table 15.2

Mean Serial Subtraction EMG
and Muscle Palpation Pain
Daily Biofeedback (N=21/23)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	.4538	.022	.4684	.014
Masseter (pre)	.5337	.008	.7057	.000
Temporalis (post)	.6600	.001	.5909	.001
Masseter (post)	.5976	.002	.5982	.001

Table 15.3

Mean Serial Subtraction EMG
and Muscle Palpation Pain
Relaxation (N=21/20)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	-.2679	.120	.0570	.406
Masseter (pre)	-.1812	.216	-.0956	.344
Temporalis (post)	-.0312	.447	.1498	.264
Masseter (post)	.2053	.186	.3888	.045

Table 15.4

Mean Serial Subtraction EMG
and Muscle Palpation Pain
Control (N=22/22)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	.1125	.309	.0036	.494
Masseter (pre)	-.2084	.176	-.2268	.155
Temporalis (post)	-.0210	.463	-.2395	.141
Masseter (post)	.1958	.191	-.2924	.093

The correlations between the EMG activity during the physical exertion stressor and the pre and posttreatment cumulative pain score were again significant for the daily biofeedback group only. Moderately strong positive correlations ranging from .6048 to .6585 were noted for the pretreatment and posttreatment variables. Pretreatment masseter EMG activity was most strongly correlated with posttreatment pain with a coefficient of .7314. The only other significant correlation from the physical exertion stress was a negative relationship (r=-.3885) between control group posttreatment masseter EMG activity and posttreatment palpation pain. The correlations between pre and posttreatment palpation pain and EMG activity recorded during the physical exertion stressor are presented in Table 16.1-16.4.

Table 16.1

Mean Physical Exertion EMG
and Muscle Palpation Pain
Weekly Biofeedback (N=23/19)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	.0708	.374	.0231	.463
Masseter (pre)	-.3467	.053	-.3576	.066
Temporalis (post)	.1915	.191	-.2285	.173
Masseter (post)	.2951	.086	-.1789	.232

Table 16.2

Mean Physical Exertion EMG
and Muscle Palpation Pain
Daily Biofeedback (N=21/23)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	.6585	.001	.6422	.001
Masseter (pre)	.6209	.002	.7314	.000
Temporalis (post)	.6967	.000	.6048	.001
Masseter (post)	.6115	.002	.6491	.000

Table 16.3

Mean Physical Exertion EMG
and Muscle Palpation Pain
Relaxation (N=21/20)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	-.1681	.233	.3338	.075
Masseter (pre)	-.1697	.231	.0533	.412
Temporalis (post)	-.1725	.227	.0309	.449
Masseter (post)	.1425	.269	.2175	.178

Table 16.4

Mean Physical Exertion EMG
and Muscle Palpation Pain
Control (N=22/22)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	-.2700	.112	-.1855	.204
Masseter (pre)	-.3193	.074	-.2179	.165
Temporalis (post)	-.1997	.186	-.2292	.152
Masseter (post)	-.1454	.259	-.3885	.037

For the post cracker mastication stressor, the daily biofeedback group again produced the only consistent correlations between pre and posttreatment EMG and palpation pain. The strongest correlation was between pretreatment temporalis muscle activity and pretreatment pain (r=.7330). The remaining correlations were all moderately strong within the .6 to .7 range.

As with the previous correlations for the other groups, the pre and posttreatment correlations for the post mastication stressor were also predominantly non-significant. The only exception was posttreatment masseter EMG and pain for the relaxation group (r=.5289). Correlations between pre and posttreatment palpation pain and EMG activity recorded during the post cracker mastication phase are presented in Tables 17.1-17.4.

Table 17.1

Mean Post Mastication EMG
and Muscle Palpation Pain
Weekly Biofeedback (N=23/19)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	.1847	.199	-.1357	.290
Masseter (pre)	-.2464	.128	-.3643	.063
Temporalis (post)	.2515	.124	-.1137	.322
Masseter (post)	.3842	.035	-.0797	.373

Table 17.2

Mean Post Mastication EMG
and Muscle Palpation Pain
Daily Biofeedback (N=21/23)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	.7330	.000	.7038	.000
Masseter (pre)	.6341	.001	.7280	.000
Temporalis (post)	.7058	.000	.6299	.001
Masseter (post)	.7087	.000	.6007	.001

Table 17.3

Mean Physical Exertion EMG
and Muscle Palpation Pain
Relaxation (N=21/20)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	-.3032	.091	.2680	.127
Masseter (pre)	-.0016	.497	.2907	.107
Temporalis (post)	-.0284	.451	.2609	.133
Masseter (post)	.3957	.038	.5289	.008

Table 17.4

Mean Physical Exertion EMG
and Muscle Palpation Pain
Control (N=22/22)

	Pain (pre)		Pain (post)	
	r	p	r	p
Temporalis (pre)	.3032	.085	.1597	.239
Masseter (pre)	-.0762	.368	-.2109	.173
Temporalis (post)	.1339	.276	-.2441	.137
Masseter (post)	.1694	.226	-.2871	.098

Symptom Chart Rating and EMG Activity

Interesting correlations were obtained between pre and posttreatment baseline temporalis and masseter EMG activity and the mean weekly symptom chart pain rating. The daily biofeedback and relaxation groups produced predominantly positive significant correlations, whereas the weekly biofeedback and control groups demonstrated generally non-significant correlations between self-report pain and pre and posttreatment EMG activity. Furthermore, while the correlations for the daily biofeedback and relaxation groups were positive, the majority of correlations in the weekly biofeedback and control groups were negative.

The largest correlations between muscle activity and mean weekly self-report pain occurred in week five for the daily biofeedback group with pretreatment baseline temporalis EMG producing a coefficient of .6102, and posttreatment temporalis activity a coefficient of .7191. It is also interesting to note that the largest correlations between pre and posttreatment EMG and self-report pain were observed the week prior to starting the treatment consisting of biofeedback and relaxation training. Additionally, only the daily biofeedback group produced significant correlations between mean weekly pain rating and posttreatment temporalis (r=.4965) and masseter (r=.5153) EMG during the final week of treatment, at the end of which, the posttreatment EMG recordings were obtained.

The relaxation group produced generally weak, but consistent positive correlations between pre and posttreatment EMG and self-report pain. In contrast with the daily biofeedback group, posttreatment relaxation group EMG only neared significance in its correlation with week 6 self-report pain (temporalis r=.3536; masseter r=.3556). With respect to the relationship between pretreatment EMG and pain during the final week of treatment, only the relaxation group produced significance (temporalis r=.4225; masseter r=.4270). For the remaining groups, pretreatment baseline EMG activity was not a significant predictor of pain during the final week of treatment. The pre and posttreatment baseline EMG and weekly pain rating correlations are presented in Tables 18.1-18.4.

Table 18.1

Baseline EMG Activity and Weekly Pain Rating
Weekly Biofeedback (N=25)

	Temporalis (pre) r	Temporalis (pre) p	Temporalis (post) r	Temporalis (post) p
Week 1	.2201	.151	-.0664	.379
Week 2	.2596	.105	-.3322	.052
Week 3	-.0536	.400	-.1254	.275
Week 4	.2504	.114	-.2276	.137
Week 5	.1050	.309	-.0784	.355
Week 6	.2041	.169	-.0973	.326
	Masseter (pre) r	**Masseter (pre) p**	**Masseter (post) r**	**Masseter (post) p**
Week 1	-.0321	.441	.1016	.318
Week 2	-.1992	.170	.0813	.350
Week 3	-.2670	.098	-.2897	.080
Week 4	-.2645	.101	-.1205	.283
Week 5	-.1645	.216	.0382	.428
Week 6	-.2193	.152	-.0582	.393

Table 18.2

Baseline EMG Activity and Weekly Pain Rating
Daily Biofeedback (N=23)

	Temporalis (pre)		Temporalis (post)	
	r	p	r	p
Week 1	.4148	.027	.5113	.006
Week 2	.3713	.044	.5199	.006
Week 3	.4987	.009	.5434	.004
Week 4	.4678	.014	.4807	.010
Week 5	.6102	.001	.7191	.000
Week 6	.0603	.400	.4965	.011
	Masseter (pre)		**Masseter (post)**	
	r	p	r	p
Week 1	.5114	.007	.4694	.012
Week 2	.4896	.010	.5402	.004
Week 3	.4527	.017	.5677	.002
Week 4	.3107	.080	.4547	.015
Week 5	.5089	.008	.6609	.000
Week 6	.1184	.309	.5153	.008

Table 18.3

Baseline EMG Activity and Weekly Pain Rating
Relaxation (N=22)

	Temporalis (pre)		Temporalis (post)	
	r	p	r	p
Week 1	.2078	.183	.2424	.145
Week 2	.4521	.017	.3909	.036
Week 3	.5243	.006	.4334	.022
Week 4	.4556	.017	.3596	.050
Week 5	.4125	.028	.3211	.073
Week 6	.4225	.025	.3536	.053

	Masseter (pre)		Masseter (post)	
	r	p	r	p
Week 1	.2518	.135	.3850	.042
Week 2	.5206	.006	.5325	.005
Week 3	.5563	.004	.4758	.013
Week 4	.3883	.037	.4198	.026
Week 5	.3948	.035	.3365	.063
Week 6	.4270	.024	.3556	.052

Table 18.4

Baseline EMG Activity and Weekly Pain Rating
Control (N=23)

	Temporalis (pre)		Temporalis (post)	
	r	p	r	p
Week 1	.3375	.058	-.1366	.267
Week 2	.4506	.015	-.1595	.234
Week 3	.2611	.114	-.1495	.248
Week 4	.1427	.258	-.3045	.079
Week 5	.3423	.059	-.0747	.371
	Masseter (pre)		**Masseter (post)**	
	r	p	r	p
Week 1	-.0694	.377	-.0767	.364
Week 2	-.1715	.217	-.2105	.168
Week 3	-.2350	.140	-.1831	.201
Week 4	-.1321	.274	-.1207	.292
Week 5	.1016	.326	-.0598	.396

EMG Activity and Subjective Improvement Rating

The correlations between pre and posttreatment baseline masseter and temporalis EMG activity and posttreatment subjective percentage improvement for the three treatment groups were predominantly negative and significant only in the daily biofeedback group. Posttreatment baseline temporalis EMG from the daily group produced a moderate negative correlation with posttreatment subjective improvement (r=-.4157), as did posttreatment masseter EMG (-.6207).

Pretreatment baseline EMG was not a significant predictor of posttreatment subjective improvement in any of the three treatment groups. The correlations between pre and posttreatment baseline EMG and subjective percentage improvement are presented in Tables 19.1-19.3.

Table 19.1

Baseline EMG and Subjective Percent Improvement Weekly Biofeedback (N=26)

	Percent Improvement	
	r	p
Temporalis (pre)	-.1524	.229
Masseter (pre)	-.0450	.414
Temporalis (post)	-.2178	.143
Masseter (post)	.1158	.287

Table 19.2

Baseline EMG and Subjective Percent Improvement Daily Biofeedback (N=25)

	Percent Improvement	
	r	p
Temporalis (pre)	-.2358	.134
Masseter (pre)	-.2435	.126
Temporalis (post)	-.4157	.019
Masseter (post)	-.6207	.000

Table 19.3

Baseline EMG and Subjective Percent
Improvement Relaxation (N=23)

	Percent Improvement r	 p
Temporalis (pre)	-.1600	.233
Masseter (pre)	-.2146	.163
Temporalis (post)	.0381	.431
Masseter (post)	.0699	.376

EMG and MMPI Scales

No consistent patterns were observed in the correlations between pre and posttreatment temporalis and masseter baseline EMG and the MMPI scale T scores. Scales 1(Hs), 2(D), and 3(Hy) were significantly correlated with pretreatment temporalis EMG activity in the control group only: 1(Hs) r=.3968, 2(D) r=.3651, and 3(Hy) r=.4521. Significant correlations were also noted for relaxation group between scale 2(D) and pretreatment EMG from the temporalis (r=.3801) and masseter (r=.4636). The pretreatment daily biofeedback group temporalis and masseter baseline EMG means were also significantly correlated with scales 1(Hs) and 3(Hy), but not with scale 2(D). No significant correlations were noted for the weekly biofeedback group on the first three clinical scales of the MMPI, although significance was observed between pretreatment masseter EMG and scales F (r=.3845) and K (r=-.3849). Details of the correlations between pre and posttreatment masseter and temporalis baseline EMG activity and the MMPI scale T scores are presented in Tables 20.1-20.4.

Table 20.1

Correlation between MMPI and Baseline EMG Weekly (N=26)

Scale	Temporalis (pre)		Temporalis (post)	
	r	p	r	p
L	.2108	.151	-.1344	.256
F	-.2964	.071	.1576	.221
K	.2176	.143	-.0578	.390
1(Hs)	-.1023	.309	.0557	.394
2(D)	-.0701	.367	.0049	.491
3(Hy)	.1258	.270	-.1701	.203
4(Pd)	-.1502	.232	-.0052	.490
5(Mf)	-.0910	.329	-.0014	.497
6(Pa)	.1324	.260	.1248	.272
7(Pt)	-.0418	.420	-.0602	.385
8(Sc)	-.2310	.128	.1258	.270
9(Ma)	-.1658	.209	-.1443	.241
0(Si)	-.1281	.266	.1446	.240

Table 20.1 (Part 2)

Scale	Masseter (pre) r	p	Masseter (post) r	p
L	-.0473	.409	.4231	.016
F	.3845	.026	.0611	.383
K	-.3849	.026	.0004	.499
1(Hs)	-.0251	.452	.0163	.468
2(D)	.0438	.416	.0698	.367
3(Hy)	-.1825	.186	.0392	.425
4(Pd)	.0762	.356	-.0180	.465
5(Ma)	.0906	.330	.1609	.216
6(Pa)	.1054	.304	.2131	.148
7(Pt)	.3182	.057	-.0563	.392
8(Sc)	.3590	.036	.0704	.366
9(Ma)	.0374	.428	.0213	.459
0(Si)	.1842	.184	-.1438	.242

Table 20.2

Correlation between MMPI and Baseline EMG
Daily (N=24)

Scale	Temporalis (pre)		Temporalis (post)	
	r	p	r	p
L	-.2510	.118	-.3007	.072
F	.1307	.271	.0962	.324
K	.2614	.109	.1220	.281
1(Hs)	.3939	.028	.3901	.027
2(D)	.0434	.420	.0134	.475
3(Hy)	.4136	.022	.3064	.068
4(Pd)	.0527	.403	-.0911	.332
5(Mf)	.2213	.149	.3363	.050
6(Pa)	-.0667	.377	-.0986	.320
7(Pt)	.0920	.335	.0780	.356
8(Sc)	.1871	.191	.1001	.317
9(Ma)	.3144	.067	.1656	.214
0(Si)	-.4080	.024	-.2925	.078

Table 20.2 (Part 2)

Scale	Masseter (pre)		Masseter (post)	
	r	p	r	p
L	-.2068	.166	-.3218	.058
F	.2986	.078	-.0456	.414
K	.1217	.286	.0338	.436
1(Hs)	.6474	.000	.2866	.082
2(D)	.3299	.058	.1367	.257
3(Hy)	.6115	.001	.1344	.261
4(Pd)	.1722	.211	-.2682	.097
5(Ma)	.0945	.330	.0661	.377
6(Pa)	.1595	.228	-.0665	.376
7(Pt)	.3952	.028	.1821	.192
8(Sc)	.4485	.014	.0984	.320
9(Ma)	.2848	.089	-.0290	.445
0(Si)	-.2628	.107	-.0738	.363

Table 20.3

Correlation between MMPI and Baseline EMG Relaxation (N=22)

Scale	Temporalis (pre) r	p	Temporalis (post) r	p
L	.1874	.202	.0774	.366
F	.1794	.212	.0580	.399
K	-.7384	.000	-.3163	.076
1(Hs)	.1928	.195	-.0039	.493
2(D)	.3801	.041	-.0541	.405
3(Hy)	-.0861	.352	-.0709	.377
4(Pd)	-.4088	.029	-.2481	.133
5(Mf)	.2094	.175	.2617	.120
6(Pa)	-.0479	.416	-.2676	.144
7(Pt)	-.0956	.336	-.2090	.175
8(Sc)	.0867	.351	-.0806	.361
9(Ma)	-.0430	.425	.0786	.364
0(Si)	.3813	.040	.0907	.344

Table 20.3 (Part 2)

Scale	Masseter (pre) r	Masseter (pre) p	Masseter (post) r	Masseter (post) p
L	.2557	.125	.1847	.205
F	.3330	.065	.5088	.008
K	-.5210	.006	-.4066	.030
1(Hs)	.2707	.112	.4741	.013
2(D)	.4636	.015	.3312	.066
3(Hy)	-.0329	.442	.1214	.295
4(Pd)	-.2441	.137	-.0703	.378
5(Ma)	.1718	.222	-.0736	.372
6(Pa)	-.0784	.364	.2011	.185
7(Pt)	.1013	.327	.2204	.162
8(Sc)	.1321	.279	.3824	.040
9(Ma)	-.0898	.346	-.1394	.268
0(Si)	.4949	.010	.6472	.001

Table 20.4

Correlation between MMPI and Baseline EMG Control (N=23)

Scale	Temporalis (pre)		Temporalis (post)	
	r	p	r	p
L	-.1200	.293	.0135	.476
F	-.1172	.297	-.3356	.059
K	.0179	.468	.2014	.178
1(Hs)	.3968	.030	.0015	.497
2(D)	.3651	.043	.0654	.383
3(Hy)	.4521	.015	-.0078	.486
4(Pd)	.4921	.009	.2013	.179
5(Mf)	-.4889	.009	-.0547	.402
6(Pa)	.2863	.093	.3703	.041
7(Pt)	.4572	.014	.1805	.205
8(Sc)	.2590	.116	.4567	.014
9(Ma)	.1613	.231	.2317	.144
0(Si)	.0066	.488	.0028	.495

Table 20.4 (Part 2)

Scale	Masseter (pre) r	Masseter (pre) p	Masseter (post) r	Masseter (post) p
L	-.0886	.344	.2905	.089
F	-.2073	.171	-.3784	.037
K	.4386	.018	.3948	.031
1(Hs)	.4297	.020	.0408	.427
2(D)	.0676	.380	-.1007	.324
3(Hy)	.2951	.086	.1304	.277
4(Pd)	.2386	.136	.3272	.064
5(Ma)	.0672	.380	-.1130	.304
6(Pa)	-.1761	.211	.1098	.309
7(Pt)	-.0221	.460	.0512	.408
8(Sc)	.0707	.374	.1738	.214
9(Ma)	.0281	.449	.1939	.227
0(Si)	-.0569	.398	-.2907	.089

Clinical Examination

Pretreatment Cumulative Pain and Palpation Sites

TMJ

Only one weak significant correlation was found between pretreatment TM-joint noise (Left Hard Tissue Grating, closing) and total palpation pain score (r=-.1837, p=.047). The remaining correlations were primarily negative, but did not approach significance. Moderate correlations were noted between the total pain score and lateral and posterior TMJ palpation. The strongest correlations with cumulative pain were found at posterior palpation of the left and right TMJs with movement, r=.6252 and .5993, respectively. Table 21 details the correlations between pretreatment temporomandibular joint palpation cumulative pain score and the individual palpation sites.

Table 21

TMJ Palpation Site and Total Pain Score
(N=87)

		TMJ pain lateral	TMJ pain posterior
No movement	(L)	.5254	.3946
	(R)	.5859	.4001
With movement	(L)	.4993	.6252
	(R)	.5179	.5993

$p<.05$

Muscles

Moderately high correlations ranging from .6020 to .7868 were noted between all muscle palpation sites and the pretreatment total palpation pain score. The left and right hyoid muscles were most strongly correlated with total palpation pain with coefficients of .7868/.7836. The lateral pterygoids also obtained moderately strong correlation coefficients of .7487/.7730, left/right, respectively. The case was similar with the left and right suboccipital region which produced coefficients of .7622/.7131. Table 22 documents the correlations between the pretreatment cumulative palpation pain scores and the individual muscles assessed.

Table 22

Muscle Pain Correlated with Cumulative Pain Score (Pretreatment) (N=87)

Muscle	Pain
Post. temporalis (L)	.6703
Post. temporalis (R)	.6338
Ant. temporalis (L)	.6700
Ant. temporalis (R)	.6257
Superficial masseter (L)	.6551
Superficial masseter (R)	.7162
Deep masseter (L)	.6020
Deep masseter (R)	.6560
Hyoid, ant. digastric (L)	.7868
Hyoid, ant. digastric (R)	.7836
Sternocleidomastoid (L)	.6343
Sternocleidomastoid (R)	.6539
Suboccipital area (L)	.7622
Suboccipital area (R)	.7131
Medial pterygoid (L)	.6329
Medial pterygoid (R)	.7200
Lateral pterygoid (L)	.7487
Lateral pterygoid (R)	.7730
Temporal tendon (L)	.6375
Temporal tendon (R)	.6329

$p<.0001$

Occlusal Characteristics

The pretreatment cumulative palpation pain scores were not significantly correlated with maximum opening or overbite. This is at variance with Gervais (1984) in which a moderate correlation was noted between degree of overbite and muscle palpation pain. Analyses of variance also revealed no significant difference in cumulative palpation pain between the three Angle occlusal classifications. The present findings suggest that occlusal characteristics are not consistently related to palpation pain in TMJ patients.

Palpation Pain and Self-report Pain

The correlations between pre and posttreatment total palpation pain and the weekly mean symptom chart ratings varied in strength from group to group. In the weekly biofeedback group, pretreatment palpation pain was significantly correlated with the mean symptom chart rating for week 1 (pretreatment baseline) only (r=.5402). Other significant correlations (r=.4083 to .5730) were observed between posttreatment pain and the weekly symptom chart ratings from weeks 2 through 6 (treatment phase).

The daily biofeedback group produced significant correlations between pretreatment clinical palpation pain and the weekly symptom chart rating for the first five weeks of the six weeks (r=.5056 to .6816), corresponding with the pretreatment baseline phase of the program. The posttreatment palpation pain scores from the daily biofeedback group did not correlate significantly with the subjective pain rating during treatment in week 6. Hence, the subjective pain rating during the treatment period was inconsistent with the posttreatment palpation pain scores, and did not predict the outcome of the posttreatment clinical examination. The relaxation group produced no significant correlations between pretreatment cumulative palpation pain and the weekly symptom chart rating. The only significant correlations for this group were weak and occurred between the posttreatment pain scores and the subjective pain rating from weeks 2, 4, and 6 (r=.3927, .4795, and .3980).

Significant correlations between pre and posttreatment cumulative palpation pain and the weekly mean symptom chart ratings were observed in the control group. Posttreatment palpation pain registered the largest correlations with the symptom chart for all five weeks of the control group program (r=.4561 to .6234). Pretreatment pain correlated significantly with the symptom chart for the first three weeks, but did not predict posttreatment palpation pain at the .05 significance level.

In summary, pretreatment palpation pain in the weekly and daily biofeedback groups was a significant predictor of the mean weekly symptom chart rating only during the pretreatment, or baseline phase of the treatment program. A similar relationship was observed for the control group. During the treatment phase of the program, only the weekly biofeedback group demonstrated a consistent relationship between the mean weekly subjective symptom chart pain ratings and the posttreatment clinical palpation pain scores. The daily biofeedback group was the only group to demonstrate a non-significant relationship between the final week of the symptom chart and the posttreatment cumulative palpation pain scores. The correlations between the pre and posttreatment cumulative palpation pain scores and the weekly mean symptom chart ratings are presented in Tables 23.1-23.4.

Table 23.1

Pre and Posttreatment Palpation Pain Correlated with Mean Weekly Pain Rating Weekly Biofeedback (N=22/18)

Week	Pain (pre)		Pain (post)	
	r	p	r	p
1	.5402	.006	.3731	.064
2	.3191	.074	.5303	.012
3	-.0111	.481	.4113	.045
4	.1293	.283	.4083	.046
5	.2172	.166	.4605	.027
6	.2107	.180	.5730	.008

Table 23.2

Pre and Posttreatment Palpation Pain Correlated with Mean Weekly Pain Rating Daily Biofeedback (N=22/18)

Week	Pain (pre)		Pain (post)	
	r	p	r	p
1	.5056	.010	.4311	.023
2	.5149	.008	.3145	.077
3	.5087	.009	.2492	.132
4	.5924	.002	.3644	.048
5	.6816	.000	.4279	.023
6	.2436	.157	-.1079	.325

Table 23.3

Pre and Posttreatment Palpation Pain
Correlated with Mean Weekly Pain Rating
Relaxation (N=20/19)

Week	Pain (pre)		Pain (post)	
	r	p	r	p
1	.2077	.197	.3191	.098
2	.2668	.128	.3927	.048
3	-.1431	.274	.1714	.241
4	.2129	.184	.4795	.019
5	.1860	.216	.3581	.066
6	.1777	.227	.3980	.046

Table 24.4

Pre and Posttreatment Palpation Pain
Correlated with Mean Weekly Pain Rating
Control (N=21/20)

Week	Pain (pre)		Pain (post)	
	r	p	r	p
1	.4270	.027	.5430	.005
2	.4191	.029	.6234	.001
3	.4384	.023	.5507	.005
4	.1707	.230	.4561	.019
5	.1981	.201	.5133	.010

Palpation Pain Rating and MMPI

Significant positive correlations were found between MMPI scales 1(Hs) and 3(Hy) and pre and posttreatment cumulative palpation pain for the daily biofeedback group (r=.4144 to .5850), and for the weekly biofeedback group at pretreatment for scale 3(Hy) (r=.3924). This is consistent with the focus upon primarily physical concerns comprising these scales. There was also a significant relationship between pretreatment pain and scale 6(Pa) for the Relaxation group (r=.4688), and also at posttreatment on scale 0(Si) (r=.4516). This is in contrast with the weekly biofeedback group which also obtained a significant, but negative, posttreatment correlation (r=-.4560) on scale 0(Si). The only other significant correlations were observed for the control group at pretreatment on scale K (r=.-4806), and at posttreatment on scale F (r=.4845). The details of the correlations between pre and posttreatment pain and the MMPI scale T scores are presented in Tables 25.1-25.4.

Table 25.1

MMPI Scores Correlated with Pre and Post Cumulative Palpation Pain Scores Weekly Biofeedback (N=23/19)

MMPI Scale	Pain (pre) r	Pain (pre) p	Pain (post) r	Pain (post) p
L	.2893	.090	.0767	.377
F	-.0421	.424	-.0763	.378
K	.1977	.183	.1567	.261
1(Hs)	.2392	.136	-.1378	.287
2(D)	-.0438	.421	-.1605	.256
3(Hy)	.3924	.032	.0855	.364
4(Pd)	.1085	.311	.0882	.360
5(Mf)	-.0829	.353	-.1762	.235
6(Pa)	.0349	.437	-.1026	.338
7(Pt)	-.1641	.227	-.3964	.046
8(Sc)	-.1291	.279	-.2778	.125
9(Ma)	.0866	.347	-.0215	.465
0(Si)	-.2894	.090	-.4560	.025

Table 25.2

**MMPI Scores Correlated with Pre
and Post Cumulative Palpation Pain Scores
Daily Biofeedback (N=21/23)**

MMPI	Pain (pre)		Pain (post)	
Scale	r	p	r	p
L	-.0257	.456	.0611	.391
F	.0033	.494	.0954	.333
K	.1209	.301	.1461	.253
1(Hs)	.4144	.031	.4726	.011
2(D)	.3141	.083	.2806	.097
3(Hy)	.4589	.018	.5850	.002
4(Pd)	-.1389	.274	.0987	.327
5(Mf)	.2659	.122	.0233	.458
6(Pa)	-.1916	.203	-.0551	.401
7(Pt)	.1093	.319	.2806	.097
8(Sc)	.0719	.378	.1378	.265
9(Ma)	.1006	.332	.1336	.272
0(Si)	-.0838	.359	-.0675	.380

Table 25.3

MMPI Scores Correlated with Pre and Post Cumulative Palpation Pain Scores Relaxation (N=20/18)

MMPI	Pain (pre)		Pain (post)	
Scale	r	p	r	p
L	.0086	.486	.1827	.234
F	.1538	.259	.1986	.215
K	.1797	.224	.1324	.300
1(Hs)	.2362	.158	.2531	.155
2(D)	-.0114	.481	.1047	.340
3(Hy)	.2402	.154	.1260	.309
4(Pd)	.3581	.061	.2762	.134
5(Mf)	.1755	.230	.1593	.264
6(Pa)	.4688	.019	.0656	.398
7(Pt)	.2894	.108	.2212	.189
8(Sc)	.2931	.105	.3685	.066
9(Ma)	-.0160	.473	-.2355	.173
0(Si)	.1479	.267	.4516	.030

Table 25.4

MMPI Scores Correlated with Pre and Post Cumulative Palpation Pain Scores Control (N=20/20)

MMPI Scale	Pain (pre) r	p	Pain (post) r	p
L	-.2764	.119	-.3361	.074
F	.2490	.145	.4845	.015
K	-.4806	.016	-.3436	.069
1(Hs)	.1988	.200	.2166	.180
2(D)	.0267	.456	.0956	.344
3(Hy)	.0504	.416	.0325	.446
4(Pd)	-.0050	.492	-.0433	.428
5(Mf)	-.3099	.092	-.2316	.163
6(Pa)	-.0915	.351	.0386	.436
7(Pt)	-.0992	.339	-.1739	.232
8(Sc)	-.1741	.231	-.2716	.123
9(Ma)	.0202	.466	.0458	.424
0(Si)	.0910	.351	-.2368	.157

Screening Questionnaire and Percentage Improvement

Pearson product correlations were obtained between the self-reported percentage improvement for the weekly biofeedback, daily biofeedback, and relaxation groups and the Screening Questionnaire. In the weekly biofeedback group, a moderately large negative correlation was found between the presence of diurnal clenching or bruxing and percentage improvement (r=-.4891). The correlations on this variable were non-significant for the daily biofeedback and relaxation groups. Chronic headaches, or neck and shoulder pains were negatively correlated with percentage improvement to a significant level in the relaxation group only (r=-.3637). In the weekly biofeedback and relaxation groups, positive responses to question 18 "Is your condition the result of an injury or accident?" were significantly correlated with self-report percentage improvement for the weekly biofeedback group (r=.4479), and for the relaxation group (r=.5452). Self-report improvement, in the relaxation group only, was positively associated with non-involvement in legal action regarding the TMJ condition (r=.4400). Finally, for the weekly biofeedback group, percentage improvement after treatment was significantly positively correlated with the time duration of the TMJ condition. The correlations between subjective percentage improvement and the screening questionnaire are presented in Table 26.

Table 26

Percentage Improvement and the Screening Questionnaire

Question	Weekly		Daily		Relaxation	
	r	p	r	p	r	p
1	-.4891	.006	-.2878	.082	.1100	.313
2	.0387	.426	.0918	.331	-.1761	.211
3	-.2356	.134	-.0345	.436	.3637	.048
4	-.0189	.464	-.2877	.082	-.2923	.106
5	.1244	.272	-.1666	.213	.1084	.311
6	.0759	.362	-	-	-	-
7	-.0473	.413	-.1225	.280	-	-
8	.0049	.491	.2309	.133	-	-
9	-	-	-.0149	.472	.3213	.067
11	-.1316	.261	.1033	.312	-.2982	.084
12	.1180	.287	-.0703	.369	.0348	.437
18	.4479	.012	.1793	.196	.5452	.004
19	.2851	.084	.0451	.415	.4400	.018
20	.4957	.021	-.3338	.088	.2981	.173

"-" indicates that a correlation could not be computed.

Posttreatment Questionnaire

Correlations were calculated between subjective percentage improvement on the posttreatment questionnaire and the frequency of home relaxation tape practice assessed by question 4. Neither the weekly nor the daily biofeedback group produced a significant correlation between subjective improvement and the frequency of home relaxation practice (r=.2400, r=.0112). For the relaxation group, however, there was a significant positive correlation between percent improvement and frequency of home relaxation practice (r=.4008).

MMPI

MMPI and Weekly Pain Symptom Rating

Significant correlations were observed within the groups between the weekly mean symptom chart rating and a number of MMPI scale T Scores. The relaxation group obtained only two significant correlations between self-report pain and MMPI scales K (r=-.4019) and 0(Si)(r=.3971) for week three of the treatment program. Although the correlations for the remaining weeks were not significant, they were all negative for scale K and positive for scale 0. In common with the relaxation group, the weekly biofeedback group also obtained a number of significant negative correlations on scale 0 for the second, fourth, and sixth weeks of treatment, with correlation coefficients ranging from -.3596 to -.4342. The weekly biofeedback group also produced significant correlations between MMPI scales 1(Hs)(r=.4660), 3(Hy)(r=.4123), and 4(Pd)(r=-.4407) during the first week corresponding with the pretreatment baseline period. The daily biofeedback and control groups produced a consistent pattern of significant moderately large correlations between self-report pain and MMPI scale 1(Hs) with coefficients ranging from .4230 to .7179. It is interesting to note that the first five weeks of the treatment program constituted an identical baseline symptom charting period for the daily biofeedback and control groups. The control group also demonstrated moderate significant correlations between self-report pain and MMPI scales 2(D) and 3(Hy) for each of the five weeks in the control period. A number of significant correlations were also noted between self-report pain and MMPI scales 4(Pd), 6(Pa), and 7(Pt) for the control group. Overall, MMPI scales 1(Hs) and 3(Hy) predicted the degree of self-report pain most consistently. The correlations between weekly mean pain rating and the MMPI scale T scores are reported in Tables 27.1-27.4.

Table 27.1

Mean Weekly Pain Rating and MMPI
Weekly Biofeedback (N=25)

	Week					
MMPI	1	2	3	4	5	6
L	.0460	.0071	-.0616	-.0406	-.1412	-.0418
F	.2605	-.0212	-.0324	-.0461	.2401	.0698
K	.0801	.2936	.3239	.3080	.0855	.1510
1	.4660*	.1419	.1819	.1777	.3821*	.2594
2	.1646	-.1111	-.0459	-.0671	.2142	.1122
3	.4123*	.2284	.1774	.2491	.2962	.3393
4	.4407*	.2091	.1892	.1166	.3145	.1570
5	.1548	.1775	.2798	.3353	.2450	.2137
6	.0418	-.2115	-.3859*	-.1600	.1050	.0541
7	.0792	-.2034	-.1126	-.0417	.0844	.0808
8	.2412	-.0575	.0123	.0907	.2172	.1750
9	.2910	.1758	.1607	.2525	.1706	.1737
0	-.2857	-.4342*	-.3275	-.3671*	-.1371	-.3596*

*p<.05

Table 27.2

Mean Weekly Pain Rating and MMPI
Daily Biofeedback (N=23)

	Week					
MMPI	1	2	3	4	5	6
L	.2375	-.0069	.0089	.0469	-.1843	.1585
F	.2215	.1655	.1384	-.0964	-.0841	.0041
K	.0899	.1207	.2144	.0591	.1657	-.0262
1	.6372**	.5753**	.5994**	.4230*	.4460*	.4413*
2	.2867	.2886	.1482	.2292	.0646	.1121
3	.5102**	.3614*	.3143	.2622	.2479	.1501
4	.2164	.0471	-.3017	-.3111	-.3068	-.2360
5	.2085	.3010	.2695	-.0499	.2695	.1406
6	.3645*	.0383	-.2381	-.1764	-.3023	-.0587
7	.5345**	.4977**	.2709	.1378	.0918	.2049
8	.4395*	.4677*	.3715*	.0476	.1477	.0789
9	.1775	.0987	.2502	.1783	.1723	.1022
0	-.0607	.0485	-.1340	-.0149	-.1410	.1221

*p<.05, **p<.01

Table 27.3

Mean Weekly Pain Rating and MMPI Relaxation (N=21)

			Week			
MMPI	1	2	3	4	5	6
L	-.3505	-.2965	-.2641	-.2027	-.2015	-.2911
F	.1715	.2179	.3645	.1694	.1556	.2376
K	-.0864	-.2430	-.4019*	-.1962	-.2816	-.2751
1	.0593	.2739	.2103	.0945	.2947	.2721
2	.0728	.0939	.3063	.0194	.0903	.0850
3	.0677	.1934	.0948	-.0161	.1718	.2128
4	.0451	-.0183	-.2274	-.2465	-.2728	-.0986
5	-.1158	.1781	-.1728	-.0177	-.0075	.0973
6	.0937	.1504	-.0926	-.1331	-.0584	.0614
7	.1312	.1600	.2073	.0407	.1169	.1920
8	.1838	.2261	.2240	.0960	.1464	.2498
9	-.0033	.0075	-.0721	-.2175	-.1246	-.0231
0	.1831	.2433	.3971*	.3445	.3342	.2614

*p<.05

Table 27.4

Mean Weekly Pain Rating and MMPI
Control (N=22)

	Week				
MMPI	1	2	3	4	5
L	-.2480	-.2533	-.3090	-.1782	-.3485
F	.1898	.3368	.3766*	.3422	.4277*
K	-.3128	-.3833*	-.3213	-.1702	-.1716
1	.5206**	.4707*	.6761***	.6274**	.7179***
2	.4469*	.5098**	.6418**	.6102**	.6845***
3	.3947*	.4285*	.6093**	.5546**	.6520**
4	.3411	.3261	.4349*	.3363	.4989*
5	-.3197	-.3871*	-.2380	-.1304	-.3000
6	.3013	.3781*	.5406**	.4326*	.5766**
7	.3025	.3311	.4091*	.4253*	.4566*
8	.1284	.1132	.3278	.2375	.3727*
9	.1157	.0979	.2143	.0681	.2133
0	.0037	-.0225	-.0653	-.0896	-.1011

*p<.05, **p<.01, ***p<.001

MMPI and Percentage Improvement

The correlations between the MMPI scale T scores and subjective percentage improvement were largely not significant at the .05 level. In the weekly biofeedback group, scale 2(D) was negatively correlated with subjective improvement to a statistically significant level (r=-.4208), as was scale 0(Si) with a coefficient of -.3512. The only other significant relationship was obtained by the relaxation group on scale 1(Hs) with a correlation of -.5115. Overall, no consistent relationship was noted between subjective percentage improvement and the MMPI scales. The correlations between percentage improvement and MMPI scale T scores are documented in Table 28.

Table 28

Percent Improvement Correlated with MMPI

MMPI Scale		Weekly (N=26)	Daily (N=25)	Relaxation (N=21)
Age	(r)	.1622	.1636	-.0790
	(p)	.214	.217	.367
L		.1729	.3247	.0593
		.199	.057	.399
F		-.0342	.0540	-.1292
		.434	.399	.288
K		.0911	.2769	.1619
		.329	.090	.242
1(Hs)		-.1888	.0329	-.5115
		.178	.438	.009
2(D)		-.4208	.1176	-.2095
		.016	.288	.181
3(Hy)		.0446	.1712	-.3666
		.414	.207	.051
4(Pd)		-.0594	.3350	-.1303
		.387	.051	.287
5(Mf)		.1626	-.0594	.0664
		.214	.389	.387
6(Pa)		-.0620	-.1136	-.0692
		.382	.294	.383
7(Pt)		-.2903	.1083	-.2115
		.075	.303	.055
8(Sc)		-.1450	.0718	-.2764
		.240	.366	.113
9(Ma)		.2436	.1869	-.2927
		.155	.185	.099
0(Si)		-.3512	.0440	-.0613
		.039	.417	.396

MBTI Frequency Distribution

The observed frequencies of the MBTI types obtained from the patient sample were tested against the expected female base rate frequencies documented in the Manual (Myers & McCaulley, 1985) for Form G using the Chi square test. The observed type table distribution was significantly different from the expected frequency distribution ($X^2 = 25.374$, $p<.05$). A z-test for individual proportions determined that the following types were under-represented in the sample: ESTJ, INTP, and ENTJ. The types ISFJ and ENFP were over-represented ($p<.05$). The type table of the patients in this study, with the expected frequencies, is presented in Table 29.

Table 29

Type Table
Expected N and %
of TMJ Patients

ISTJ E=9.18 9.77%	ISFJ* E=9.68 10.30%	INFJ E=4.48 4.77%	INTJ E=3.76 4.00%
ISTP E=2.50 2.67%	ISFP E=4.01 4.27%	INFP E=5.94 6.32%	INTP* E=3.00 3.20%
ESTP E=2.61 2.78%	ESFP E=5.38 5.73%	ENFP* E=9.21 9.80%	ENTP E=3.86 4.11%
ESTJ* E=9.46 10.07%	ESFJ E=10.02 10.66%	ENFJ E=5.99 6.38%	ENTJ* E=4.85 5.17%

*Significantly different from base rate, p<.05

The over-representation of the ISFJ and ENFP types in the sample may provide some clues regarding the personality structure of temporomandibular pain-dysfunction patients. ISFJ individuals can be described as conscientious, loyal, devoted, service-minded, meticulous, and responsible (Hirsh & Kummerow, 1990). They are motivated by a high sense of duty and, possibly more than other types, are prone to being taken advantage of in marriage, family, or job situations (Kroeger & Thuesen, 1988). Kroeger and Thuesen indicate further that ISFJs' sense of responsibility and duty to others permeates every aspect of their lives, with the result that "relaxation can come only when all work is completed and. . . it rarely is" (p. 220). ISFJs may complain bitterly about their excessive demands and responsibilities, but there is nothing they would rather be doing. When an attempt is made to relieve the ISFJ'S burden, she is hurt and beset by guilt (Kroeger & Thuesen, 1988).

The ENFP type is creative, enthusiastic, spontaneous, energetic, and restless (Hirsh & Kummerow, 1990). The enthusiasm and energy of ENFPs, combined with a need to be affirmed and accepted by others, leads them to easily overexert and overextend their physical and psychological resources. They can work themselves to the point of exhaustion in their search for approval (Kroeger & Thuesen, 1988). As with ISFJs, relaxation is difficult and requires effort for the ENFP individual. As described by Kroeger & Thuesen, "ENFPs go in fits and starts, and so when they become excited, they lose all sense of time, physical needs, and anything else. They follow their enthusiasm until totally fatigued, then collapse. As a result, relaxation, unless part of a creative adventure, may take a backseat, sometimes even at the expense of the ENFP's physical well-being" (p. 259).

While some overlapping of personality characteristics is seen between the MBTI types, the particular combination of characteristics observed in the ISFJ and ENFP types emphasize a high degree of energy expenditure in combination with a lack of due care and concern for personal needs, especially those involving leisure, self-nurturing, and relaxation. Consequently, individuals with ISFJ and ENFP preferences could be more prone to developing stress-related disorders than the other MBTI types. Indeed, the descriptions given above are similar to those forwarded by other investigators who characterized temporomandibular patients as rigid, hypernormal, matriarchal, dominant, responsible, generous, emotionally repressed, anxious, and tense (Kydd, 1959;

Lupton, 1966, 1969; Heloe, Heiberg, & Krogstad, 1980).

The role of the inferior function in the MBTI theory is also an interesting dimension to consider in the analysis of the TMJ patient types presented above. In type theory the four-letter code represents the dynamic relationship between the individual's preferred way of obtaining energy, attending to incoming information, making decisions, and lifestyle. The middle two letters refer to the dominant and auxiliary functions, those preferences which take the lead the individual's interaction with the internal or external world. The tertiary function is the opposite to the auxiliary on the preference scale, and the inferior is the opposite to the dominant function. The inferior function is not characteristic of the individual but manifests itself in times of stress or illness, in sum, when the individual is not acting like himself (Hirsh & Kummerow, 1990).

Intuition is the inferior function of the ISFJ individual. Whereas this individual normally relies on the dominant Sensing function, when under stress she will use Intuition excessively resulting in a tendency toward pessimism and negativity. She will also perceive herself as being stuck in a rut and see no possible way of alleviating the situation (Hirsh & Kummerow, 1990). The ENFP, while under the influence of the inferior

Sensing function, will tend to become obsessed or preoccupied with unimportant facts and details. Sensory pursuits may predominate with an overindulgence in food, exercise, or potentially, an excessive focus upon physical or emotional symptoms (Hirsh & Kummerow, 1990).

The two MBTI types described above may provide a means of understanding those TMJ patients who manifest primarily symptoms of depression, and those who produce a more hypochondriacal or hysterical presentation. This analysis of the temporomandibular pain-dysfunction patient, if pursued in greater depth with larger samples, may provide additional insight into the personality characteristics which predispose or accompany the disorder.

D. Summary

The patients in the present study demonstrated more TMJ related symptoms than the subjects assessed in Gervais (1984). This was seen in higher cumulative palpation pain scores, self-report symptoms, and EMG activity from the temporalis and masseter musculature. At baseline and during presentation of the physical and mental stressors, all subjects responded with a significant increase in masticatory muscle activity. The temporalis consistently demonstrated significantly greater activity than did the masseter. All groups demonstrated a statistically significant reduction in EMG activity following the treatment program or control period. There was no significant difference between the groups. Similarly, all groups registered a significant reduction in palpation pain and TMJ noise scores following the treatment or control period.

The groups did not differ significantly in posttreatment degree of improvement, as measured by the pre and posttreatment clinical examinations, although the weekly biofeedback group had the greatest percentage change in palpation pain with a 62% reduction as compared to the daily biofeedback group with an 18% decline in pain. The relaxation and control groups each recorded a 34% reduction in palpation pain at the posttreatment assessment. The daily pain chart did not register a significant change over the duration of the study, regardless of group membership. The weekly biofeedback group reported the greatest subjective improvement over the course of the treatment program, with a 45% improvement as compared to daily biofeedback and relaxation groups with 26% and 27% subjective improvement ratings, respectively. The differences between the groups in self-report improvement were statistically significant at the .05 level.

The correlations between temporalis and masseter EMG activity and pre and posttreatment palpation pain at baseline, and for the stressors, were significant only in the daily biofeedback group. Although the weekly biofeedback, relaxation, and control groups produced some significant correlations, there was no consistent relationship between pre and posttreatment EMG activity and pre and posttreatment palpation pain. Significant correlations between pre and posttreatment EMG activity and the weekly mean pain ratings from the daily symptom chart were observed predominantly for the daily biofeedback group, and, to a lesser extent in the relaxation group, but generally not for the weekly biofeedback or control groups. The correlations between posttreatment EMG activity and subjective percentage improvement in the three treatment groups were significant only in the daily biofeedback group.

No consistent relationship between the MMPI scale T scores and pre and posttreatment EMG activity was noted, although scales 1(Hs) and 3(Hy) produced significant correlations in the daily biofeedback and control groups. Scale 2(D) was also significantly correlated with EMG activity in the relaxation and control groups. No significant correlations with these scales were found for the weekly biofeedback group.

From the pretreatment clinical examination findings, primarily non-significant correlations were found between the cumulative palpation pain scores and

the individual TMJ noise scores. Conversely, moderate significant correlations were noted between TMJ palpation pain scores and cumulative palpation pain. Similarly, moderately high correlations were observed between the individual muscle palpation scores and the cumulative palpation scores. The left and right hyoid muscles demonstrated the strongest correlations with total palpation pain, followed by the left lateral pterygoid and the left suboccipital area.
Occlusal characteristics such as range of maximum opening and degree of overbite were not significantly correlated with pretreatment cumulative palpation pain.

Pretreatment cumulative palpation pain and weekly mean symptom chart ratings were significantly correlated in the weekly and daily biofeedback groups during the pretreatment or baseline phase of the program. This was also observed in the control group, but not in the relaxation group. The correlations between posttreatment pain and the weekly mean symptom chart ratings obtained during the treatment phase were significant for the weekly biofeedback and relaxation groups, but not for the daily biofeedback group.

A number of significant correlations were found between the cumulative palpation pain scores and MMPI scales 1(Hs) and 3(Hy) in the weekly and daily biofeedback groups. In general, however, there was no consistent relationship across the groups between the MMPI scale T scores and cumulative palpation pain. Similarly, subjective percent improvement was essentially not significantly correlated with the MMPI scale T scores.

The weekly mean pain symptom chart ratings were significantly correlated with a number of MMPI scale T scores. Most notable were consistent positive correlations between self-report pain and MMPI scale 1(Hs) for the daily biofeedback and control groups. Scales 2(D) and 3(Hy) also received a number of significant correlations with the mean weekly self-report pain ratings. The relaxation group demonstrated the least degree of correlation between MMPI scale scores and mean weekly self-report pain.

There were no consistent significant correlations between subjective percent improvement and the Screening Questionnaire, although a moderate negative correlation was noted for bruxing or clenching in the weekly biofeedback group. Subjective improvement was also positively correlated with the presence of a trauma or accident related TMJ condition for the weekly biofeedback and relaxation groups. Involvement in legal action was negatively related to subjective improvement in the relaxation group. The length of duration of TMJ symptoms was positively correlated with subjective improvement in the weekly biofeedback group.

There was no significant correlation between frequency of home relaxation practice with the relaxation tape and subjective improvement for the weekly and daily biofeedback groups. A moderate positive correlation between these variables was found for the relaxation group.

Finally, the MBTI type frequency distribution of the patients in the study was significantly different from the expected female base rate. The types ESTJ, INFP, and ENTJ were under-represented in the sample, whereas the types ISFJ and ENFP were over-represented.

V. DISCUSSION

A. Major Findings

The present study was designed to compare the effectiveness of two biofeedback training schedules, progressive relaxation training, and a no-treatment control condition in reducing the signs and symptoms of temporomandibular pain and dysfunction. The specific treatment outcome variables in all groups consisted of pre and posttreatment temporalis and masseter EMG activity, clinical palpation pain, and daily pain symptom ratings. Subjective posttreatment percentage improvement was also obtained in the three treatment groups.

The results of this study show that all groups registered significant reductions in EMG activity ($p<.05$) in the posttreatment repeated measures. There were no statistically significant between groups differences in mean EMG activity, although the weekly biofeedback group tended to demonstrate less variability in posttreatment EMG levels.

Similarly, all groups demonstrated a significant time effect with respect to clinical palpation pain and TMJ noise scores. Again, there was no statistical difference between the groups, despite the weekly biofeedback group obtaining a 62.14% reduction in palpation pain in contrast to the daily biofeedback group (18.31%), the relaxation group (34.68%), and the control group (34.39%).

As regards subjective posttreatment percentage improvement, there was a statistically significant difference in outcome ($p<.05$) between the three treatment groups with the weekly biofeedback group reporting a mean 45.38% improvement in temporomandibular pain-dysfunction symptoms, in contrast to 26.20% for the daily biofeedback group, and 27.38% for the relaxation group. Conversely, however, the weekly mean pain rating derived from the daily self-report pain chart showed no significant change for any of the groups over the course of the study.

Hence, although two of the four posttreatment dependent measures of treatment outcome demonstrated a significant time effect, they failed to achieve between groups significance. The daily symptom chart registered neither a significant time nor group effect. This left only the self-report percentage improvement to discriminate between the treatment groups, suggesting a significant treatment effect for the weekly biofeedback group.

On the basis of these results, the five week program of weekly biofeedback training could not be clearly deemed superior to either the same program provided on five consecutive days, home-based relaxation training only, and a no-treatment control condition in reducing the signs and symptoms of temporomandibular pain and dysfunction. Indeed, the daily biofeedback, relaxation and control conditions appear to have produced approximately statistically equivalent benefits.

No consistent pattern emerged in the correlations between the amount of baseline or stress-related EMG activity in the temporalis and masseter muscles and self-report or clinically assessed palpation pain. With the exception of the daily biofeedback group, the correlations between pain and EMG activity were largely non-significant. Hence, the psychophysiologic theory of temporomandibular disorders, which maintains that pain and other signs and symptoms of dysfunction are related to stress-induced masticatory muscle hyperactivity, and targets reduction of this activity as a primary treatment goal, cannot be corroborated. This suggests that the pain experience for TMJ patients is dependent upon a number of factors other than specific neuromuscular hyperactivity. The large degree of variability in EMG activity and clinical symptoms within the sample of patients also suggests that individual differences in the interaction between muscular hyperactivity and other non-specific factors in the expression of pain and other symptoms are also important considerations.

B. Relationship to Previous Research

The literature reporting the application of biofeedback training in temporomandibular disorders is mixed in its assessment of treatment outcome. In general, the use of biofeedback as an effective therapeutic modality is endorsed, although as Mealiea and McGlynn (1978) indicate, there is as yet no strong experimental evidence to support this conclusion. The results of the present study are also mixed with only one of the four dependent outcome measures, subjective percentage improvement, showing a significant treatment effect for the weekly biofeedback protocol. In light of these predominantly non-significant findings, the primary conclusion of this study is at variance with much of the literature in stating that there is no firm evidence that EMG biofeedback training is more effective than relaxation training or no treatment in managing temporomandibular dysfunction.

There are a number of possible explanations for the failure of the present study to corroborate the findings of other investigators. Some relate to shortcomings peculiar to this study, while others stem from the methodological inadequacies of previous experimental and clinical studies.

One of the major difficulties in assessing the claims of the literature regarding the effectiveness of biofeedback in temporomandibular disorders is the paucity of sufficiently rigorous research protocols. In the experimental method, the values of dependent variables are compared in the presence of independent variable values (Mahoney, 1987). If the manipulation of the independent variable(s)--in clinical outcome research, the provision of treatment--is associated with a predictable and replicable change in the dependent variables, a treatment effect is considered to have occurred. There are, however, a number of threats to the validity of the purported treatment effect. Some of these as described by Mahoney (1978) include: inadequate sample size, or one that is unrepresentative of the population to which the conclusions are to be applied; lack of random assignment to the various treatment conditions; poorly specified independent variable(s); inadequate standardization, description, or assessment of how the independent variable was implemented; inadequate control of auxiliary variables; inadequate replication of the cause-effect relationship; poor selection or assessment of dependent variables; and conclusions or interpretations not logically derived from the experimental procedures. Rigorous experimental methodology endeavors to augment the internal and external validity of any observed treatment effect by using designs intended to control or compensate for these potential threats to validity and minimize their influence. The use of large sample sizes, random assignment to treatment groups, no-treatment control and placebo groups, and comparative treatment conditions are among the most common means of strengthening experimental designs (Mahoney, 1978; Horan, 1980; Parloff, 1986; Kazdin, 1986; Basham, 1986).

An examination of the research methodology employed in the clinical and experimental reports reviewed earlier reveals that many of the reports used single-case or small sample designs using an XO or OXO format (O=observation, X=treatment). Mahoney (1978) rates these types of designs, respectively, as "extremely weak" and "weak." Furthermore, of the 21 studies documenting the application of biofeedback in treating temporomandibular disorders, only six incorporated control group designs.

Accordingly, the findings and conclusions of much of the biofeedback/TMJ literature to date are of questionable internal and external validity as there is no way to reasonably determine whether the observed changes were due to the treatment, or to chance and other non-specific factors. An example of this type of ambiguity of outcome can be seen in Dahlström, Carlsson, Gale, and Jansson (1985) who compared 20 mandibular dysfunction patients and 20 healthy controls with respect to stress-related changes in masseter EMG activity before and after receiving six sessions of biofeedback. Predictably, the patient group demonstrated significantly higher stress-induced EMG levels than the control subjects at pretreatment. Following biofeedback training, the patients demonstrated significant reductions in stress-related EMG activity, whereas the control subjects were unchanged. There was consequently no significant difference in stress response between the two groups. The authors concluded that biofeedback training helped the patients to control their muscle activity and responsiveness to stress with an associated decrease in symptomatology.

Superficially, the above conclusion appears to corroborate the effectiveness of biofeedback training in temporomandibular disorders. The present study also found a significant reduction in stress-related EMG activity following biofeedback training. In contrast with Dahlström et al. (1985), however, the untreated patient control group also registered significantly reduced EMG at retest. This suggests that EMG activity in patients reduces whether or not biofeedback training is actually provided. Furthermore, the control group used by Dahlström et al., being composed of healthy subjects rather than equivalent patients, is by definition not a true control group, a shortcoming which weakens the validity of the purported treatment effect considerably. The results of the present study suggest that, if the earlier study would have incorporated a patient control group, both groups might also have shown a significant reduction in EMG activity regardless of treatment. Thus, the findings of Dahlström et al. could reflect habituation, placebo effects, or simply the normal cyclical fluctuations of mandibular dysfunction symptoms over time, rather than true treatment effects.

The present study, in contrast to most of the investigations reported in the literature, employed a considerably stronger design incorporating a relatively large patient sample, a patient control group, an extended pretreatment baseline period in the daily biofeedback group, and a number of well-defined pre and posttreatment dependent measures. Despite being limited by its inability to control for participation or observation effects, the design of this study is rated as "generally adequate" according to Mahoney's (1978) criteria. Consequently, although the results of this study do not conform to the general findings of much of the literature, the relative superiority of its experimental design strengthens the validity of the obtained findings and conclusions.

Another distinction between the present study and the bulk of the clinical and experimental literature lies in the nature of the dependent measures used to assess treatment outcome. Whereas this project used four dependent measures, two objective (experimentally blind clinical examinations and EMG) and two subjective (daily symptom charting and self-report improvement), the majority of reports base their conclusions solely upon patient self-report and/or EMG activity (Mealiea & McGlynn, 1987). A notable exception is the work of Stenn, Mothersill, and Brooke (1979) who incorporated self-report, blind physician rating, and masseter EMG. The use of multiple dependent measures increases the confidence with which one can make conclusions regarding the nature and magnitude of the experiment or treatment effect (Mahoney, 1978). From this perspective, the results of the present study might be considered stronger and more definitive than those of other investigators. However, as in all clinical or experimental studies, this project also has a number of limitations and shortcomings. These will be discussed in the following pages.

C. Limitations and Delimitations

The limitations and delimitations of the present study will be presented under two categories: 1. diagnostic issues and, 2. research design and methodology issues.

Diagnostic Issues

As was discussed in the literature review, temporomandibular disorders have traditionally been classified under two major diagnostic categories: a. disorders of primarily organic etiology, and b. functional or non-organic disorders (MPD). Similarity among patients' problems or diagnoses is essential for valid experimental research (Kazdin, 1978) hence, the present study was originally intended to target patients belonging to the latter diagnostic category. The distinctions separating the two categories are, however, neither universally accepted in theory, nor clearly observed in clinical practice. For this reason the range of TMJ disorders and myofascial pain dysfunction were considered as part of a continuum of polarized but invariably overlapping clinical signs and symptoms.

While this philosophical orientation may, on the one hand, have been convenient in facilitating admittance into the study for the large number of patients required, it may also have introduced an unforeseen degree of diagnostic heterogeneity, and hence, variability, into the patient sample. This variance could have been better controlled by adhering more strictly to diagnostic criteria such as those described by Bell (1986). For the purposes of the present study, the patients sought would have received primary diagnoses within the general category I. Masticatory Muscle Disorders. This category contains conditions related to protective muscle splinting, spasm, and muscle inflammation. In addition to diagnoses in this category, patients classified under section II. Disc-interference Disorders, would also have been common.

On the other hand, the practical reality of identifying and referring an adequate number of suitable patients according to relatively narrow diagnostic criteria poses a considerable challenge for the clinical practitioner faced with a multitude of patients, all of whom are demanding effective treatment. Consequently, it appears that the patients ultimately referred to this study manifested a considerable range of mandibular dysfunction signs and symptoms. As such they did not constitute a homogenous diagnostic group within the MPD classification as originally intended, but rather, represented a cross section of temporomandibular pain dysfunction disorders, a limitation which may in part explain some of the inconclusive findings of the present study.

A related limitation was the lack of a standardized procedure to train the participating dentists in administering the pre and posttreatment clinical examinations. A videotape describing the procedure would have helped to strengthen inter-rater reliability and may have decreased the amount of variability in patient symptoms. Nonetheless, as the majority of patients were referred from the practice of Dr. N. Thomas, and from a relatively small number of dentists specialized in the treatment of temporomandibular disorders, and as the pre and posttreatment clinical examinations were conducted by the same individual, the reliability of the assessments was considered adequate.

The use of a well-accepted standard clinical assessment procedure such as that developed by Helkimo (1974), would also have helped in making comparisons between the patients treated in this study and those described in the literature. The clinical examination procedure applied in this project was identical to that used in Gervais (1984) and was retained in order to facilitate comparisons of patient characteristics between the two studies.

The lack of a posttreatment administration of the MMPI led to some difficulty in interpreting the correlations between the personality measures and other variables related to treatment outcome, notably EMG activity, daily symptom chart pain rating, and posttreatment subjective percentage improvement. In the absence of posttreatment MMPI data, it is not possible to ascertain whether posttreatment improvement was a function of personality change or other specific treatment effects. The most that can be determined from the present findings is that a simple reduction in EMG activity does not appear to be the primary active effect underlying the success of biofeedback training. Personality changes over the course of treatment, particularly with respect to MMPI scales 1(Hs), 2(D), 3(Hy), and possibly 7(Pt), are likely related to improvement, however, no definitive statement can be made on the basis of the present findings.

Another limitation in this study was the inability to control for other physical or psychological variables not immediately related to the referring diagnosis of temporomandibular pain dysfunction. These uncontrolled variables, while presumably randomly distributed between the groups, may have also interfered with the efficacy of the treatment and obscured the outcome. An example of this is a patient who met the diagnostic criteria for the study, but whose symptoms were of secondary importance to what was generally medically accepted as a clear case of classical conversion hysteria accompanied by paralysis of an arm and aphonia. Despite good compliance, this patient, as would be expected, did not report a significant improvement in her condition. Other examples of interference due to uncontrolled psychological variables include a number of patients with previous psychiatric histories and evidence of ongoing psychopathology. Providing that a reasonable commitment to the study could be obtained, these individuals were not excluded. The presumably active, and possibly chronic, psychopathology in these patients was likely a major impediment to their successful outcome.

A substantial number of patients whose condition was related to motor vehicle accidents or some other form of recent physical trauma were also referred to the study. In these patients, jaw pain and related symptoms were usually only one of a number of presenting problems including neck, shoulder, and back pain. Although the treatment provided may have benefitted these patients specifically with respect to their temporomandibular disorders, the global effects of the treatment were not assessed, nor were any specific procedures implemented to control the potential interference of other concurrent physical symptoms.

Furthermore, of the patients referred secondary to a motor vehicle accident, head injury with resulting cognitive and emotional effects was also suspected in approximately 50% of cases. The results of neuropsychological assessment subsequent to the study did confirm central nervous system injury in a number of these patients. Evidently, the presence of cognitive or emotional effects due to brain injury would likely also influence the treatment outcome in these patients.

The lack of discrimination between patients suffering from acute or intermittent pain and those with chronic intractable pain is another limitation in the present study. As Keefe and Hoelscher (1987) maintain, there is an important difference between these two categories of patients. Those whose pain is acute or intermittent will invariably present with different psychological and behavioral characteristics than patients suffering from chronic intractable pain. Both types of patients were represented in the sample which included temporomandibular symptoms ranging in duration from as little as one year, to the case of one elderly patient whose symptoms had been chronic for fifty years. The mean duration of symptoms in the present study was 7.95 years with a standard deviation of 9.34 years. This would characterize the patient sample as generally chronic, however, there was considerable variation in their symptom history which would also have had an effect in their response to treatment. As Keefe and

Hoelscher (1987) note, biofeedback and other self-regulation therapies are less likely to be effective when the condition is chronic. Conversely, the present study found non-significant correlations between duration of symptoms and subjective improvement for all groups except the weekly biofeedback condition which produced a positive correlation on these variables.

Research Design/Methodology Issues

The present study contained a number of limitations related to research design and methodology. The lack of true random assignment of patients into the treatment and control groups is the most obvious. The purpose of random assignment in research is to minimize bias and ensure that auxiliary variables are evenly distributed between the experimental groups (Mahoney, 1978). Mahoney notes, however, that true random distributions on all variables are impossible with a finite sample and, consequently, any conclusions derived from the results must be cautious. In the present project patients were assigned to groups sequentially, in order of their presentation for the study. The sequential assignment of patients to one of four groups could be considered a pseudo-random process, which, while not perfect, permitted the study to be conducted within the practical bounds of a concurrently functioning clinical practice by assuring balanced numbers in each treatment group. One potential disadvantage which discouraged the use of true random assignment was the possibility of exceeding the facility treatment capacity by having unbalanced numbers of patients from the biofeedback groups enter treatment simultaneously. Thus, the sequential assignment method was selected as an acceptable compromise between true randomization and a non-random approach.

Although an analysis of covariance demonstrated that the four groups were not statistically different with respect to pre and posttreatment cumulative palpation pain and noise scores, the apparent differences between the control group and the three treatment groups in the clinical examination findings are an obvious example of potential skewing of data resulting from the lack of true random assignment to groups. Whereas the three treatment groups were equivalent in terms of pretreatment clinical palpation pain symptoms, the control group demonstrated approximately 50% less symptom severity. This unexpected pretreatment difference between the control group and the other groups may indeed have been due to the sequential assignment process used, however, the relatively uniform clinical characteristics in the treatment groups suggests otherwise. An alternative explanation for this discrepancy is also possible.

The ethical guidelines governing research with human subjects state:

> With due concern for the limitation of their comprehension, potential participants should be given a full and frank explanation of the purpose of the research and a full explanation of procedures to be followed, together with a careful estimate of the risks and benefits (Social Science & Humanities Research Council of Canada, 1981, p. 3).

In keeping with this ethical requirement, all subjects were given a brief written description of the study in addition to any discussion during telephone or office enquiries. At the intake appointment the design of the study and the treatment procedures, including the four groups, were again reviewed. Potential participants were clearly informed that assignment to treatment group would proceed according to the procedure described above. Only after the participants consented to accept membership in any group was the actual group assignment made, and the treatment agreement executed.

Difficulties arose in the case of certain patients who, for various reasons, wished to receive one specific treatment rather than another, or who were unable to commit themselves to requirements of the particular treatment schedule. As Kazdin (1978) indicates, in outpatient treatment research, patients' preference for one form of treatment over another poses a serious threat to randomization, one which may ultimately render true randomization unfeasible. In this study, the patients either accepted their group assignment, or withdrew from the study. The control group was particularly problematic in that the five week no-treatment control period was generally the least desirable option. This was compounded by the fact that patients would have to bear the inconvenience, and occasionally, the expense of two clinical examinations before receiving any treatment. For a number of potential control patients these requirements were simply unacceptable and they withdrew from the study.

Another difficult situation in the assignment of the control group participants was dealing with patients in acute pain. Invariably, when informed that they might have to wait as long as five weeks before commencing treatment, and furthermore, refrain from commencing any other concurrent treatment, many of these patients were unable to commit themselves to the study and withdrew. Others entered the study, but withdrew or did not satisfactorily complete the requirements. The remaining patients who agreed to participate as control subjects were likely those experiencing the least amount of acute or intractable pain or dysfunction, and were therefore most inclined to tolerate the five week control period. The discrepancies between the pain scores of the control group and the three treatment groups may well have arisen from this self-selection process. However, since research participants have the right to informed consent and the right to withdraw at any time, these inconsistencies could not easily have been avoided, even with the use of a more rigorous randomization procedure. As Hatch (1987) writes:

> . . . when patients ask questions, request a recommendation, or express a preference for one treatment over another they must be treated honestly and fairly, even if it means failing to recruit the patient for the study. Informed consent allows the patient to decide the balance between his or her individual need for treatment and his or her obligation or desire to contribute something to society (p. 362).

The apparent heterogeneity of the patient sample also indicates another potential limitation in the study. If the patients in this study were indeed relatively heterogenous with respect to temporomandibular pain-dysfunction symptoms, it may not be reasonable to seek specific therapeutic effects following the standardized treatment procedures. Either the diagnosis and selection of patients must conform to precise and commonly accepted criteria, or the treatment offered should be adaptable in order to meet individual needs.

As was discussed in the review of the literature, there is little consensus over the definition, diagnosis, and treatment of temporomandibular disorders. For this reason, there is an inherent difficulty in conducting research which targets a specific subset of temporomandibular patients. This will invariably lead to differing degrees of heterogeneity within the patient sample. Unfortunately, by applying unvarying treatment protocols to heterogenous patient samples, specific treatment effects may be overlooked and potentially useful therapies may be deemed ineffective. Keefe and Hoelscher (1987) describe this dilemma:

> In controlled research studies, treatment procedures are typically standardized, and every patient is provided with a highly similar training protocol. It may well be that better results are obtained when individually tailored programs are used. Future research should compare individually tailored biofeedback treatment approaches to standard approaches for different chronic pain syndromes (p. 245).

Thus, another limitation of the present study derives directly from the empirical research tradition which requires comparison of standard treatments across groups of presumably homogenous patients. These patients, in reality, may not only demonstrate considerable random variability, but also diagnostic heterogeneity which may obscure the effects of the treatment and lead to inconclusive results.

More precise information regarding the specific treatment effects might have been obtained through the use of a placebo group receiving bogus biofeedback or some other equally credible, but inert treatment. This would help control for effects of attention, patient expectancy, and participation in research. As Mahoney (1978) indicates, therapeutic change may occur from the mere participation in any experience which is presented as treatment. In the present study, therefore, there may have been a variety of patient expectations, regardless of group membership, which may have influenced the outcome of therapy. Furthermore, the expectations held by the referring specialists may have influenced the findings of the posttreatment clinical examinations. This may partially explain the significant posttreatment clinical improvement noted in the control group. Alternately, the improvement in the control group may also be related to the normal fluctuations of temporomandibular symptoms over time.

In addition to patient expectations and attention factors, the possibility of a placebo effect cannot be ignored. Strong placebo effects were reported by Greene and Laskin (1972), and Goodman, Greene, and Laskin (1976) who found that groups of patients improved significantly when treated only with a non-occluding, placebo splint. Green and Laskin (1971) and Laskin and Green (1972) also reported substantial improvement in two groups of patients who received placebo medication. The daily symptom charting required of all participants may have served as an attention/demand factor as well as a form of placebo in some patients.

The lack of verifiable controls over concurrent, and potentially competing treatment factors is another limitation in the present study (Kazdin, 1978). At the outset of the study, the initial intention had been to recruit only patients who had not previously been treated, or for whom treatment had proved ineffectual or had stabilized the condition. In reality, most patients had received some form of prior intervention, usually in the form of splint or physical therapy, and were referred to the study for what they perceived as an adjunctive treatment. While the majority of patients agreed not to alter their usage of occlusal splints, and defer starting a new or modified splint only after the conclusion of the study, no strict control of this compliance variable was enforced. Similarly, patients were asked to not change any previously stable medication regimen. Unfortunately, compliance in this area was again impossible to regulate. Hence, it could be argued that some of the significant changes observed over the course of the study are due to these confounding non-controlled variables. Alternately, the group assignment procedure, while not truly random, should have been sufficiently robust to assure a reasonably uniform distribution between the groups of any uncontrolled concurrent treatment variables.

Finally, the lack of a long-term posttreatment follow-up period is a shortcoming which limits the conclusions that can be drawn regarding maintenance of treatment benefits or generalization of therapeutic effects following the biofeedback and/or relaxation programs. Most of the posttreatment clinical examinations were obtained within three weeks of the final treatment session, and hence the treatment effects observed should most appropriately be considered relatively short-term. No assessment of the long-term treatment outcome is possible with the data at hand, however, a number of patients did spontaneously contact the office a number of months posttreatment to report continued benefits. A long-term follow-up enquiry could have been conducted by telephone at six, twelve, and eighteen month intervals, however, this was not practical given the time constraints of the present study.

D. The Research Questions

The present study was conducted to answer two primary questions related to the use of EMG biofeedback training in the treatment of temporomandibular pain and dysfunction:

1. Is EMG biofeedback more effective than relaxation training or no treatment in reducing the signs and symptoms of temporomandibular pain-dysfunction?
2. Is the biofeedback training protocol more effective when presented according to a daily schedule as opposed to once weekly?

Secondary correlational questions were formulated as follows:

3. What is the correlation between clinical palpation pain EMG activity?
4. What is the correlation between subjective self-report pain and EMG activity?
5. What is the correlation between EMG activity and subjective percentage improvement?
6. What is the correlation between MMPI scales and EMG activity, clinically assessed temporomandibular pain and dysfunction, self-report pain, and subjective percentage improvement following treatment?

Question One: Is EMG biofeedback more effective than relaxation training or no treatment in reducing the signs and symptoms of temporomandibular pain-dysfunction?

The answer to this question involves considering the signs and symptoms of mandibular dysfunction from two perspectives:

a. objective clinical signs and symptoms;

b. subjective symptoms.

Objective Clinical Signs and Symptoms

The objective clinical signs and symptoms of temporomandibular dysfunction were those obtained in the pre and posttreatment clinical examinations, as well as the EMG recordings from the baseline and stressors obtained pre and posttreatment. For the sake of clarity, the determination of outcome will be based on the change between the pre and posttreatment measures.

EMG Activity

As indicated in Tables 10.1-10.4, the EMG activity for the temporalis and masseter muscles at baseline and during the stressors was significantly less at posttreatment than at pretreatment ($p<.0001$). There was, however, no significant difference between the groups ($p>.05$). Hence, it appears that biofeedback training was no more effective in reducing EMG activity from the temporalis and masseter muscles, than either relaxation training or no treatment.

From a clinical perspective, however, the weekly biofeedback group generally demonstrated the least amount of variability in EMG activity at posttreatment as evidenced by the consistently smaller standard deviations. This suggests that, while the four groups may not have been significantly different from one another at posttreatment, the weekly biofeedback group appears to have learned more stable and reliable control over temporalis and masseter EMG activity following the biofeedback training period.

Noise and Palpation Scores

As was the case with the EMG activity, significant reductions in TMJ noise ($p<.05$) and palpation pain ($p<.001$) across all groups were registered at the posttreatment examinations. Again, however, statistical significance between the groups was not achieved ($p>.05$). This lack of statistical significance may be related to inadequate power in the present study. A post hoc power analysis using a variety of tests resulted in power coefficients in the .50 range. This indicates that the probability of making a Type II error, that is, not finding a difference that is there, is approximately 50%. The power of this study could have been increased by reducing the variability of the various dependent measures used, as well as by incorporating a larger sample of patients in each of the four treatment conditions. Indeed, the post hoc power analysis indicated that, given the variability of the dependent measures used, a total sample size of 170 patients would have produced a power coefficient of .83. With greater power, there would have been decreased probability of missing significant differences between the groups on the treatment outcome measures, and the observed posttreatment differences between the groups would have been significant at the .05 level. With such findings, the hypothesized efficacy of biofeedback in the management of temporomandibular disorders might have been corroborated.

Subjective Symptoms

Two measures of subjective symptoms were recorded in this study: a. self-report daily symptom chart, and b. posttreatment subjective percentage improvement rating.

Symptom Chart

The analysis of the daily symptom chart revealed that no significant changes in pain intensity were recorded over the duration of the study by any of the groups. Nor were there any significant differences between the groups. Thus, although definite clinical improvement was noted at the posttreatment clinical examinations, the patients' subjective day-to-day assessment of their condition was not consistent with the degree of objective clinical improvement. On the one hand, it could be argued that biofeedback training is no more effective than relaxation or no treatment in altering patients' daily self-report pain symptom rating. On the other hand, it could also be argued that the reliability of patient daily self-report is low and does not accurately measure the degree of therapeutic change.

Posttreatment Subjective Percentage Improvement

In contrast to the findings of the daily symptom chart, the treatment groups were significantly different ($p<.05$) with respect to self-report percentage improvement as indicated on the posttreatment questionnaire. The weekly biofeedback group obtained a mean improvement of 45.38% as compared to the relaxation group which registered 27.38%, and the daily biofeedback group which rated itself 26.20% improved. On the basis of this posttreatment subjective self-report, the five session program of weekly biofeedback training was superior to relaxation training with respect to improving the subjective symptoms of temporomandibular dysfunction.

The above conclusion can, however, be questioned from the perspective of placebo and demand effects.

Horan (1980) argues that the placebo phenomenon is not a function of what is actually done to subjects, but rather, what they believe is being done to them. In as much as patients believe they are receiving a credible and effective treatment, they will expect certain benefits. These expectations for improvement must be controlled in treatment outcome research before the validity of any emergent treatment effects can be determined. Accordingly, any investigation of treatment outcome must attempt to generate equivalent patient expectations for improvement for each of the treatment or control conditions administered. Without equivalent patient expectations, the influence of the placebo phenomenon in the measures of treatment outcome cannot be confidently ruled out.

In the present study, all patients were informed that they would be receiving one of two well-recognized treatments for temporomandibular disorders, biofeedback and relaxation training. Although the patients generally accepted their assigned treatment without difficulty, the daily biofeedback and relaxation group patients could have felt less actively involved in their treatment. For the daily biofeedback patients, there was the initial five-week baseline period, essentially identical to the control group condition. During this period, patients may well have developed a sense of abandonment or distance from the treatment which would have lowered treatment expectations. Similarly, the relaxation patients, not receiving any regular in-office contact, may have also, experienced a perceived lack of involvement in the treatment process which would have also lowered their expectations for improvement. The weekly biofeedback group, conversely, was provided with the most regular opportunity for direct involvement in treatment. This could reasonably be expected to have increased not only their perception of the treatment's efficacy, but also strengthened their expectation for improvement.

As placebo or demand factors were not specifically controlled in the present study, the between groups differences in subjective improvement ratings should be interpreted with caution, particularly as there were no significant differences in the other treatment outcome measures.

Question Two: Is the biofeedback training protocol more effective when presented according to a daily schedule as opposed to once weekly?

As with Question One above, the assessment of treatment outcome will consider the signs and symptoms of mandibular dysfunction from two perspectives:

a. objective clinical signs and symptoms;

b. subjective symptoms.

Objective Clinical Signs and Symptoms

EMG Activity and Rate of Learning Control

At posttreatment, both the weekly and daily biofeedback groups demonstrated significant reductions in EMG activity, during the baseline and stressors. As there was no statistically significant difference in EMG activity between either of the weekly and daily biofeedback groups and the relaxation or control groups, it is difficult to make any conclusive statements regarding rate of learning EMG control. On the basis of these results, neither biofeedback schedule is clearly more effective than relaxation alone or no treatment in training patients to voluntarily regulate EMG activity. Notwithstanding the reductions in EMG observed with both biofeedback schedules, as well as with the relaxation and control conditions, the consistently smaller standard deviations observed at posttreatment in the weekly biofeedback group suggest that the weekly schedule was more effective in reducing the variability of EMG activity than was the daily schedule. This may indicate that the weekly biofeedback schedule promoted more stable learned control of temporalis and masseter EMG activity than did the daily schedule, resulting in less random or irrelevant EMG activity in the weekly group, although the mean EMG levels may not have been significantly different from those of the daily, relaxation, or control groups.

The narrower range of variability noted in the EMG levels of the weekly biofeedback group, as compared to the daily biofeedback group, suggests that learning to control EMG activity by means of biofeedback is more effective when the training sessions are scheduled at wider intervals. This would appear to provide more time between sessions to develop an awareness of one's internal responses to stress and the cues triggering learned relaxation or self-regulation responses. With a biofeedback training schedule extending over the course of a number of weeks, there is potential for more effective generalization of self-regulation responses as the learning would occur over a presumably wider range of life experiences than would be encountered in a period of five consecutive days of training. Furthermore, the extended rather than compressed or short-term practice of concurrent relaxation techniques, as in the progressive relaxation used in this study, would also appear to enhance the learning of voluntary control over muscle activity attained in the biofeedback training and promote generalization of self-regulation skills into daily life.

Noise and Palpation Scores

Both biofeedback groups registered significant decreases in noise and palpation pain at the posttreatment clinical examination. Due to the large variability in palpation scores, the differences between the weekly and daily biofeedback group were not statistically significant. Although the 61.14% reduction in palpation pain obtained in the weekly biofeedback group appears clinically significant when contrasted with the 18.31% reduction in pain symptoms found in the daily biofeedback group, and suggests that a weekly biofeedback protocol is superior to a daily schedule in reducing the muscular pain associated with temporomandibular dysfunction, this conclusion cannot be supported statistically.

The joint noise scores showed a 34.25% reduction in the weekly group, but no change in the daily group. While the change in joint noise at posttreatment was statistically significant for all groups, the lack of a significant between groups effect suggests that the weekly biofeedback program was not more effective in reducing joint sounds than the equivalent program following a daily schedule.

Subjective Symptoms

The subjective symptoms monitored included the daily symptom chart and the posttreatment percentage improvement ratings. These will be discussed individually.

Symptom Chart

The conclusions presented above regarding the daily symptom chart are applicable in this discussion of the effectiveness of daily versus weekly biofeedback training in the management of temporomandibular dysfunction and pain. As there were no significant changes in self-report pain from the first to the final week of the treatment program for any of the groups, no firm conclusions can be drawn from the results of this measure of treatment outcome. From one perspective, it could be stated that weekly and daily biofeedback are equally ineffective in reducing the degree of daily self-report pain. From another perspective, the lack of significant change over the course of the treatment program calls into question the reliability of patient self-report pain rating, particularly in light of the significant changes in clinical findings and the posttreatment percentage improvement ratings.

Posttreatment Subjective Percentage Improvement

As regards posttreatment subjective percentage improvement, there was a statistically significant difference between the weekly and daily biofeedback groups. The weekly group reported a mean improvement of 45.38% compared to 26.20% for the daily biofeedback group. On the basis of this measure of treatment outcome, the five sessions of biofeedback training, provided on a weekly schedule, lead to greater subjective improvement in temporomandibular symptoms than an equivalent number of biofeedback sessions provided on a daily basis. A comparison of the percentage improvement scores for the weekly and daily biofeedback groups suggests that the superiority of weekly biofeedback is not only statistically, but clinically significant. This conclusion must be tempered, however, in consideration of possible placebo and demand effects as discussed above.

In summary, the clinical measures of treatment outcome revealed significant improvement in all patients regardless of group membership. Of the subjective measures, no change was noted in daily self-report ratings, but a significant treatment effect emerged for the weekly biofeedback group. With the clinical measures carrying relatively greater weight than the subjective measures, the efficacy of biofeedback training in reducing the signs and symptoms of temporomandibular dysfunction and pain could not be unequivocally corroborated. The findings regarding the effectiveness of weekly versus daily biofeedback in reducing EMG activity in the temporalis and masseter muscles are also inconclusive. Nonetheless, the weekly biofeedback schedule appeared to have more effectively promoted learned control of temporalis and masseter activity than did the daily biofeedback group.

Question Three: What is the correlation between clinical palpation pain and EMG activity?

The correlations between clinical palpation pain and EMG activity, pre and posttreatment, were consistently positive and significant only for the daily biofeedback group as reported in tables 14.1-17.4. This corroborates the hypothesized link between increased masticatory muscle activity and pain which is central to the psychophysiologic theory of temporomandibular pain and dysfunction. Conversely, however, the correlations for the other groups were essentially non-significant, or where significant, were not consistently positive or negative. In this instance, the psychophysiologic theory is not corroborated by the present findings. An example from the weekly biofeedback group is the significant negative correlation (r=-.4632) between pretreatment serial subtraction EMG and palpation pain. The correlation between the same variables for the daily biofeedback group produced a coefficient of .5337. Thus, as regards the daily biofeedback group only, the present results corroborate the relationship between temporomandibular pain and increased temporalis and masseter EMG activity. Consequently, treatment approaches such as biofeedback which seek to teach conscious relaxation or self-regulation of excessive muscle activity receive cautious support as logical therapeutic modalities which should be effective in managing temporomandibular dysfunction disorders.

The inconsistent correlations from the weekly biofeedback, relaxation, and control groups suggest, alternatively, that factors other than elevated temporalis and masseter activity are also associated with the clinically observed palpation pain. Whereas inconsistent correlations between posttreatment palpation pain and EMG activity might have been expected between the groups due to the varying treatment protocols, the pretreatment correlations were expected to be relatively similar in size and direction. The observed inconsistencies may be due to a number of factors including insufficient sample size, inadequate randomization, diagnostic heterogeneity, patient expectancy, and other cognitive factors. If further study of the relationship between EMG activity and palpation pain can control for such potential confounding factors, more definitive conclusions should be possible.

The lack of significant and consistent correlations between palpation pain and EMG activity has also been noted by Dahlström, Carlsson, Gale, and Jansson (1984) who conclude that there is little support for a simplistic model of biofeedback training in which clinical outcome corresponds directly to the patient's ability to reduce muscular overactivity. The inconsistent correlations noted in the present study between clinical findings and EMG activity in the weekly biofeedback, relaxation, and control groups may also be partially explained by Jacobson's (1967) observation that pain does not diminish proportionally with muscle tension, but only when total relaxation is achieved. While this non-linear relationship between pain and EMG activity may have been operating in all the groups, other factors, as described above, may have played a more significant role in the outcome of the weekly biofeedback, relaxation, and control groups; but exerted less of an influence in the daily biofeedback group.

Question Four: What is the correlation between subjective self-report pain and EMG activity?

In order to simplify the statistical analysis, the daily pain scores were collapsed to provide mean weekly pain scores. No consistent pattern of correlations was obtained between the groups with respect to pre and posttreatment EMG activity and the weekly mean symptom chart pain ratings. The daily biofeedback and the relaxation group produced predominantly positive significant correlations between these variables as compared to the weekly biofeedback and control groups whose correlations were primarily negative and non-significant.

As was indicated in Chapter 4, the largest correlations between muscle activity and mean weekly self-report pain occurred in week five for the daily biofeedback group. The fifth week for the daily biofeedback group coincided with the start of treatment following five weeks of baseline pain symptom charting. These findings could be interpreted from the perspective of perceived versus actual control as described by Biedermann, McGhie, Monga, and Shanks (1987). Biedermann et al. found that the amount of perceived and actual success back patients experienced in controlling EMG activity was not a factor in treatment effectiveness. The authors hypothesized that the effectiveness of treatment came from the patients' developing a biomechanical understanding of their back problems rather than a more threatening psychological view of their condition. The patients then developed coping strategies based upon their new biomechanical understanding of back pain.

In the context of the present results, it could be argued that the five week baseline period prior to starting biofeedback served to sensitize the daily biofeedback group to their pain and masticatory muscle activity. If this occurred, the daily biofeedback patients may have been rendered more prone to developing a direct association between their pain, the level of muscle tension in the jaw, and their control over it. This would constitute the first stage in developing a biomechanical understanding of their temporomandibular condition. Furthermore, as the five hours of biofeedback training were compressed into five consecutive days within one week, the patients in this group may have tended to interpret their pain or other subjective symptoms primarily in reference to the EMG information they were receiving daily, thus reinforcing the biomechanical perspective. Ultimately, the daily biofeedback group may have used the biofeedback information to an inordinate degree in assessing their progress. If this were the case, lack of perceived success in meeting the biofeedback training goals of reducing and balancing EMG activity in the four muscles would be interpreted as failure, and no significant difference in subjective pain would be anticipated, or consequently, reported. To complicate matters, the five day biofeedback program may not have allowed sufficient time for patients to develop alternative coping strategies based upon their biomechanical understanding of the problem, as Biedermann et al. suggest happened in their group of patients. Hence, the EMG-pain correlations in the daily biofeedback group could be interpreted as indicating that the first stage in developing a biomechanical understanding was met, but not the second requiring the formulation of coping strategies specific to this view of the problem. In this scenario, relatively large positive correlations, such as those observed in the daily biofeedback group, could be expected between EMG activity and self-report pain.

After the daily biofeedback group, the relaxation group produced the largest correlations between self-report pain and EMG activity. Pretreatment temporalis and masseter EMG correlated significantly with self-report pain for all five treatment weeks. A curious finding was that the pain chart from the pretreatment baseline week did not correlate significantly with pretreatment EMG. Near significant correlations were obtained between posttreatment temporalis and masseter EMG and self-report pain for the final week of relaxation training, however, the pretreatment EMG levels were stronger predictors of the pain chart ratings. These findings suggest that, for the relaxation group, pretreatment EMG activity was a significant predictor of daily pain rating throughout the relaxation training phase of the program, but not during the baseline portion. It appears that, as in the daily biofeedback group, the treatment phase promoted a greater awareness of masticatory muscle activity, which then became more directly associated with the perception of pain than in the weekly biofeedback or control groups. Hence, the level of pretreatment EMG activity, while not initially identified as a pain factor, became more central to the pain experience of the relaxation group as the relaxation training progressed.

The essentially non-significant correlations between pre and posttreatment temporalis and masseter EMG activity and the daily symptom chart in the weekly and control groups suggests that elevated EMG activity was not strongly related to the subjective experience of pain for either group. In the control group, there was evidently no treatment-related increase in masticatory muscle awareness as in the other groups, hence, the perception of pain was likely influenced by a variety of factors apart from any awareness of muscle tension. Similarly, the weekly biofeedback group, by learning muscle awareness and relaxation skills over a five week period may not have developed as much of a direct association between muscle tension and pain as did the daily biofeedback and relaxation groups. Additionally, over the extended period of biofeedback training the weekly group may also have learned to associate other physiologic or emotional cues with pain, thus making EMG activity a relatively insignificant contributor to the overall pain experience.

In summary, the answer to Question Four is inconclusive. The results of this study suggest that EMG activity is significantly, although only weakly to moderately, correlated with subjective self-report daily pain rating in treatment contexts involving relaxation training or a condensed biofeedback training program. In a standard biofeedback program involving weekly training sessions, EMG activity does not appear to be a significant predictor of self-report daily pain rating.

Question Five: What is the correlation between EMG activity and subjective percentage improvement?

As with the other correlations between EMG activity and the treatment outcome variables discussed above, the significance of the correlations between EMG levels and subjective percentage improvement varied from group to group. The weekly biofeedback and relaxation groups produced no significant correlations on these variables, while for the daily biofeedback group, posttreatment baseline EMG activity and subjective percentage improvement were moderately negatively correlated.

In keeping with the discussion of Question Four, these findings suggest that the levels of temporalis and masseter EMG activity are not important predictors of subjective improvement in either relaxation training or standard weekly biofeedback. Conversely, in a biofeedback protocol involving daily training, a decrease in EMG activity is significantly related to subjective improvement. As discussed above, it would again appear that the daily biofeedback patients developed a close association between EMG activity and pain. With the daily biofeedback practice, the patients in this group were more likely to remember their EMG "scores" from day to day, and would consequently rate their success in training in terms of whether or not they developed conscious voluntary control of the EMG activity and were able to match or improve upon the previous day's performance. If success in reducing EMG activity were the primary personal criterion of treatment success, a relatively strong association between EMG levels and the pain experience could be expected.

For the weekly biofeedback group, the elapsed time between sessions would facilitate the development of other personal measures of treatment success beyond the actual EMG feedback. These could include the daily pain rating, experiences of success with the home relaxation practice, the natural day-to-day variations in symptom severity, as well as the normal healing process over a five-week treatment period. Consequently, the weekly biofeedback group might be less prone to associating improvement primarily with success in controlling or reducing EMG levels from week to week. It follows, therefore, that the correlations between EMG activity and subjective percentage improvement would be relatively small.

The relaxation group treatment did not involve working with any form of direct EMG feedback. Consequently, the measures of success would be more closely associated with subjective, internal relaxation cues rather than with objective indicators of muscle activity as in the biofeedback groups. As in the weekly biofeedback group, the five week duration of treatment would also promote the development of other treatment outcome criteria as well as expose the patients to the natural fluctuations in their condition. Subjective improvement would therefore be related to a number of factors among which posttreatment EMG activity would not necessarily be significant. Indeed, of the three treatment groups, the relaxation group produced the smallest correlation between posttreatment EMG levels and subjective percentage improvement.

In summary, there appears to be a significant correlation between a decrease in EMG activity and subjective improvement where the biofeedback protocol involves daily training. In a more standard biofeedback protocol based on weekly sessions, or in relaxation therapy, the level of posttreatment EMG activity does not emerge as a significant predictor of subjective improvement.

Question Six: What is the correlation between MMPI scales and EMG activity, clinically assessed temporomandibular pain and dysfunction, self-report pain, and subjective percentage improvement following treatment?

EMG Activity

No consistent correlations were observed between the groups with respect to the MMPI scales and EMG activity. MMPI scales 1(Hs) and 3(Hy) were significantly positively correlated with pretreatment temporalis EMG in the daily biofeedback and control groups. This suggests that the EMG activity in these groups increased with the number of items endorsed relating to concern with physical functioning, particularly headaches and chronic fatigue and pain (1Hs), in addition to general denial of psychological problems or social discomfort (3Hy). The relaxation group, alternately, produced the only significant posttreatment EMG correlation with MMPI scale 1. The weekly biofeedback group displayed no significant relationship between EMG activity and scales 1 or 3 of the MMPI. The inconsistencies of the correlations between these two MMPI scales suggests, on the one hand, that there are no specific personality characteristics which predict temporalis or masseter EMG activity across all temporomandibular dysfunction patients. On the other hand, the significant correlations which were obtained may reflect the association between elevated EMG activity and specific physical and psychological concerns in patients, who, due to faulty randomization, may not have been equally distributed among the groups. The remaining MMPI scales produced a number of significant correlations with EMG activity, but again, no consistent pattern emerged across the groups.

In summary, the strength of the correlations between the MMPI scales and EMG activity in this sample of temporomandibular pain and dysfunction patients partially supports a predictive relationship between scales 1(Hs) and 3(Hy) and EMG activity from the temporalis and masseter muscles. This relationship is not consistent throughout the sample and cannot be generalized to all temporomandibular patients. Although there were a number of other significant correlations between EMG levels and MMPI scales, these also were not observed uniformly across the groups and cannot be used to make specific predictions regarding personality variables and the degree of EMG activity.

Palpation Pain

MMPI scales 1(Hs) and 3(Hy) were significantly correlated with both pre and posttreatment palpation pain only in the daily biofeedback group. Pretreatment pain and MMPI scale 3(Hy) were also significantly related in the weekly biofeedback group. The relaxation and control groups produced no significant correlations on these scales. One possible interpretation for these findings is that the daily biofeedback group registered the least change in palpation pain at the posttreatment examination (18.31%) compared to the weekly biofeedback group which showed the greatest degree of improvement (62.14%). The pretreatment personality variables were therefore moderately strong predictors of posttreatment outcome in the daily biofeedback group which, overall, registered the least amount of symptomatic change. It would have been informative to have re-administered the MMPI following the treatment in order to determine if clinical improvement was significantly associated with changes in personality variables.

As was discussed above, the daily group may have developed a stronger focus upon objective physical cues such as EMG levels as determiners of treatment success than did the other groups. If this were indeed the case, elevated scores on MMPI scales 1(Hs) and 3(Hy) would have reflected the pretreatment preoccupation of the daily group with physical symptoms, symptoms which, combined with the external cues provided by the biofeedback, constituted primary subjective criteria of outcome. The observation that the correlations between EMG and MMPI 1(Hs) and 3(Hy) are larger at posttreatment than at pretreatment for the daily biofeedback group also suggests that their focus upon physical symptoms and denial of psychological distress remained constant, if not increased, over the course of the treatment program.

In the weekly biofeedback group, posttreatment pain was not predicted by either MMPI scales 1(HS) or 3(Hy). Neither in the relaxation group, nor in the control group, did MMPI scales 1(Hs) and 3(Hy) predict pre or posttreatment palpation pain. In conclusion, there is some evidence to associate pretreatment clinically assessed pain with elevations on MMPI scales 1(Hs) and 3(HY), however, this is not found across all groups in the present study. Consequently, the presence of elevated scores in these MMPI scales cannot be reliably used to predict the severity of palpation pain in a clinical examination situation.

Self-report Pain

With the exception of the relaxation group, MMPI scale 1(Hs) and 3(Hy) were significantly correlated with self-report pain during week one of the project. The daily biofeedback and control groups continued to show significant correlations on these variables throughout the five week program, while the weekly biofeedback group achieved significance only on Week 5 for scale 1(Hs). The relaxation group produced no significant correlations between MMPI 1(Hs) or 3(Hy) and self-report pain for any week of treatment.

None of the groups received treatment during the first week, and only the weekly biofeedback and relaxation groups started treatment during the second week. The predominantly non-significant correlations with self-report pain on these MMPI scales during the treatment phases of these two groups suggests that excessive preoccupation with physical symptoms or denial of psychological problems, apparent at the outset of the study, were not significant predictors of the pain experience throughout the treatment study. In the weekly biofeedback group, the onset of treatment appears to coincide with a change in the relationship between personality characteristics and pain. This may indicate that self-report pain either diminished in intensity, was defined in terms different from the symptoms comprising the items for MMPI scales 1(Hs) and 3(Hy), or that personality characteristics were changing.

For the daily biofeedback group, it would appear that the relationship between personality characteristics, physical symptoms, and reported pain was not significantly modified either by the five week baseline period, or by the actual treatment received. In the relaxation group patients, the MMPI scales 1(Hs) and 3(Hy) were not significant predictors of self-report pain at any time during the treatment study. Finally, control group self-report pain throughout the study was predicted to a moderate level by MMPI scales 1(Hs) and 3(Hy). This also suggests that the interaction between personality factors and self-report pain, in the absence of treatment, did not change significantly over the five week control period.

The control group MMPI scale 2(D) was also significantly correlated with self-report pain to a moderate level for each week of the treatment study. Indeed the magnitude of the correlation between these variables generally increased as the weeks progressed. This suggests that in the control group, pretreatment elevations on a measure of depression not only predicted pretreatment self-report pain, but continued to predict the degree of pain reported in the absence of treatment over a five week period.

The control group, and the daily biofeedback group to a lesser extent, also produced a pattern of consistent positive significant correlations between scales 6(Pa) and 7(Pt) and self-report pain. Elevated scores on these scales indicate suspiciousness, oversensitivity, and anxiety, characteristics which may have been made more prominent by the requirement that both groups complete a five week no-treatment baseline period, during which no significant change in pain was recorded. For some participants, unfamiliar with research methodology, the control or baseline conditions may have been perceived as a form of manipulation or deception on the part of the experimenter, or, at best, an unnecessary prolongation of pain by delaying the start of treatment.

The correlations between scales 6(Pa) and 7(Pt) and self-report pain for the weekly and relaxation groups were essentially non-significant with the exception of a single negative correlation on 6(Pa) at Week 3 for the weekly biofeedback group. The finding of non-significant correlations on these variables may in part be related to treatment anticipation and expectations in the weekly biofeedback and relaxation groups who knew that their treatment would commence following a relatively brief baseline period. It could be argued that these patients had less opportunity to exacerbate any pretreatment suspiciousness, oversensitivity, or anxiety as they entered a valid treatment program without undue delay.

In summary, pretreatment MMPI scores on scales 1(Hs), 2(D), and 3(Hy) appear to be moderate predictors of pre and posttest self-report pain where treatment is not provided. Where biofeedback or relaxation training is provided, the value of these MMPI scales in predicting posttreatment pain is questionable. They may, however, be moderately associated with pretreatment self-report pain.

Subjective Percentage Improvement

The correlations between MMPI scales and subjective posttreatment percentage improvement were largely non-significant for the three treatment groups. Nonetheless, pretreatment MMPI scale 2(D) in the weekly biofeedback group produced a significant negative correlation with posttreatment improvement. A moderate negative correlation was also noted on scale 1(Hs) for the relaxation group. This suggests that posttreatment subjective improvement increases as the amount of pretreatment clinical depression and somatic preoccupation decreases. However, since significant correlations on these variables were not found across the three treatment groups, only limited support is given for the ability of MMPI scales 1(Hs) and 2(D) to predict posttreatment outcome.

E. Directions for Further Research

Future research in the application of biofeedback training in the treatment of temporomandibular disorders should attempt to provide greater definition in at least three areas:

1. Diagnostic criteria and patient selection,
2. Biofeedback assessment and treatment protocols,
3. Identification of the active effects of biofeedback training.

Diagnostic Criteria and Patient Selection

In future research, every attempt should be made to decrease the degree of diagnostic heterogeneity which appears to have been a factor in this study, as well as in previous research. As Mealiea and McGlynn (1987) suggest, the commonly used descriptions of patients as generic TMJ or MPD lack precision, and can obscure active effects and treatment outcome. Greater diagnostic precision could be accomplished by adhering to a well-defined classification system such as Bell (1986) in the pre and posttreatment clinical examinations. If a uniform patient sample could not readily be identified for referral, then several subclassifications of patients could be made within the study, based on the detailed clinical examination findings. Although it is practically unrealistic to expect temporomandibular disorder patients to fall exclusively into one specific diagnostic classification, a primary diagnosis could be made on the basis of the most prominent class of symptoms, followed by secondary diagnoses for other contributing symptoms. In the analysis of treatment outcome, patients within the same primary diagnostic classification could then be contrasted with one another rather than with patients falling under a different primary diagnostic classification.

Another consideration in further research is the distinction between acute and chronic pain patients. In the writer's experience, the majority of temporomandibular patients referred for biofeedback training tend to be chronic sufferers for whom routine splint or other dental management has been ineffective or inadequate. The therapeutic outcome of biofeedback training might be substantially different with a group of acute or other non-chronic patients such as those who generally obtain adequate relief from standard dental management. Additionally, further studies contrasting biofeedback with other types of dental, physical, or psychological therapies with groups of patients, well-matched regarding acute and chronic pain, may help to determine the comparative efficacy of each approach.

Many patients develop temporomandibular disorders following motor vehicle accidents or some other physical trauma to the head, neck, and jaw. The proportion of such patients in the present study was 38.8%. In the author's clinical practice, the frequency of patients with accident-related TMJ problems is at least comparable, if not higher than that obtained in this study. A history of head and neck trauma can precipitate not only temporomandibular dysfunction, but also a variety of cognitive, affective, vestibular, and auditory symptoms. While these symptoms may be commonly subsumed under the generic label of "TMJ", they can also indicate the presence of cerebral injury or damage to the inner ear, quite distinct from the temporomandibular disorder itself.

As was indicated above, a number of the patients in the study were found to have some degree of neuropsychological impairment upon subsequent testing. Future research should, particularly when dealing with motor vehicle accident patients, incorporate a neuropsychological screening battery in order to distinguish between those patients whose symptoms are clearly TMJ-related, and those who may be manifesting signs of cognitive impairment as well as mandibular dysfunction. As much of the biofeedback treatment with temporomandibular patients is conducted by psychologists, routine screening of post-accident patients for neuropsychological symptoms is not only justifiable and relatively simple to conduct, but may also provide a wealth of diagnostic information that would not normally be obtained from the regular dental or medical investigations.

As regards the auditory and vestibular symptoms commonly observed in temporomandibular patients, especially following a motor vehicle accident or head injury, the recently defined perilymph fistula syndrome (PLFS) (Grimm, Hemenway, Lebray, & Black, 1989), provides important insight into the etiology and refractory nature of some common "TMJ" symptoms including headache, tinnitus, fullness of the ears, ear pain, hearing loss, and disequilibrium. The patients described by Grimm et al. were chronic, with a mean duration of 75.8 weeks (S.D.= 84.5). As in temporomandibular disorders, the patients were predominantly female (66.3%) with a mean age of 35.3 years (S.D.= 10.8). Follow-up studies of untreated or previously undiagnosed cases of PLFS indicate that the condition can persist for years. Some of the inconclusive treatment outcome results reported in the biofeedback/TMJ literature, as well as in the general TMJ literature, may be partially due to unrecognized PLFS symptoms in a significant proportion of the unsuccessfully treated TMJ patients. Future treatment outcome research in temporomandibular disorders should be aware of PLFS as a potential confounding variable and either exclude these patients, or place them within a separate subcategory or treatment group.

Biofeedback Assessment and Treatment Protocols

The development of broader EMG assessment protocols should also be a goal of future research in biofeedback applications to temporomandibular disorders. Most clinical biofeedback reports have typically used the masseter as the target muscle for assessment and training. However, as suggested in Moss and Garrett (1984), and discussed in Gervais (1989), as well as in the present study, simultaneous multi-site recordings can provide important diagnostic information regarding muscle balance, hyperactivity, and hypoactivity. Treatment outcome reports of multi-site EMG assessment and biofeedback training have been lacking in the literature for two principle reasons. The first relates to equipment limitations, and the second to what may be considered an excessive reliance on clinical tradition.

Until relatively recently, most biofeedback instruments were generally capable of one or two channel recording and display, hence most clinical reports have documented single, or at most, dual site assessment or training. In recent years the use of microcomputers has revolutionized the process of data collection and integration of multiple feedback signals into a workable display for training purposes. The present study was conducted using a four channel biofeedback system, permitting the simultaneous integration EMG activity from the masseter and temporalis muscles. There are now a number of relatively affordable units on the market capable of recording and displaying up to 16 channels of physiological information simultaneously. Assessment protocols using these EMG capabilities should help to provide a more precise perspective on the individual patient's condition, and lead to more effective treatment planning. Furthermore, in the investigation of stress response and temporomandibular disorders, multi-channel assessment capabilities would provide the opportunity to use other modalities such as peripheral skin temperature and galvanic skin response (GSR) to obtain added insight into the autonomic characteristics of patients.

In addition to using multiple recording sites, static and dynamic assessments of mandibular movement should also be obtained. Static and dynamic assessment protocols have been used in biofeedback approaches to back pain with significant success (Donaldson, 1989). Some caution in using dynamic assessment procedures in temporomandibular disorders is recommended as jaw movements can be influenced by a number of factors including disc interference disorders, degenerative and inflammatory process, ankylosis and pseudoankylosis, as well as abnormal muscle function (Bell 1986). Precise differential diagnosis of these various conditions is essential, therefore, when conducting and interpreting an EMG assessment, particularly with respect to dynamic movements.

The predominant use of the masseter as a standard or default biofeedback assessment or training site with temporomandibular patients, is easily understood given the frequent observations of symptoms such as bruxing, clenching, preauricular pain, stiffness, and limited opening. Masseter biofeedback, usually from the painful side, is a credible procedure based on the assumption that pain is related to excessive EMG activity in the target site. Conversely, clinical experience using the multi-site assessment techniques reported in this study has found that greater EMG activity contralateral to the site of pain is frequently observed (Gervais, Fitzsimmons, & Thomas, 1989). Furthermore, the report of Gervais et al., and the finding in the present study, that EMG activity in the temporalis was significantly higher than in the masseter ($p<.0001$), challenges the continued use of the masseter as a default EMG feedback site. Indeed, this finding is consistent with the unilateral or bilateral temple region headaches reported by many temporomandibular dysfunction patients and may prove to be a generally more effective training site for some patients. In summary, further research should attempt to obtain EMG recordings from multiple sites before the training sites are selected.

As in Janata (1986), a bilateral balancing, or four site equalization approach as used in the present study, could be used in biofeedback training. The efficacy of this type of biofeedback training protocol with back pain patients has recently been demonstrated by Donaldson (1989), and holds considerable promise for temporomandibular biofeedback therapy. Future research should investigate the effectiveness of multi-site balancing protocols in contrast to the conventional single-site relaxation approaches.

Identification of Active Effects

Finally, the identification of specific treatment effects could be facilitated by the routine administration or monitoring of multiple dependent measures. As Nelson (1981) indicates, multiple dependent measures should be used as there is no one true measure of a client's problem. In the present study, the pre and posttreatment clinical examinations, EMG recordings, MMPI, and the daily symptom chart were the primary dependent measures. Additional psychological and physiological measures of anxiety, locus of control, stress rating and stress response might have contributed to a more complete picture of the patient sample, and helped to isolate specific treatment effects in subgroups of patients or in individual cases. In particular, the over or under-representation of a number of MBTI types in the present study may provide additional insight into the personality characteristics of temporomandibular patients, and factors which may be related to positive treatment outcome.

It should also be recognized that a particular treatment may have a variety of non-specific effects. Consequently, the use of multiple measures is also important to determine whether the treatment is having a specific effect in the area intended, or whether it is producing side effects in other dimensions (Nelson, 1981). A good example of this effect in the present study would have been to monitor the elevation of the MMPI scales or some measure of self-efficacy or anxiety to note any changes occurring concurrently with the biofeedback training.

As was discussed above, the specific treatment effects responsible for the degree of improvement observed in this study do not appear to be primarily related to reduced muscle activity acheived through EMG biofeedback and/or relaxation training. The fact remains, however, that certain patients in each of the treatment groups reported important subjective and clinical gains, although from the perspective of between groups contrasts, the superiority of one treatment modality over the others could not be established. It may be hypothesized that treatment success in biofeedback, and other self-regulation therapies, derives from a number of factors such as increased perception of control over muscle activity leading to an altered understanding of the nature of the pain and dysfunction (Hijzen, Slangen, & Van Houweligen, 1986; Biedermann, McGhie, Monga, & Shanks, 1987), motivation for change (Dahlström, Carlsson, Gale, & Jansson, 1984), and self-efficacy beliefs and expectations (Dolce, 1987). The significance of these and other proposed alternative treatment effects should be examined empirically in future research by administering pre and posttreatment dependent measures which will specifically isolate these effects. In this way a greater understanding of the mechanisms underlying the purported efficacy of biofeedback therapies in the treatment of temporomandibular disorders may be attained.

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Appendix A

BIOFEEDBACK OUTCOME STUDY

Information for dentists

Thank you for participating in this treatment outcome study. In brief, the basic requirements of the research design are as follows:

1. Female patients whose diagnosis is mandibular dysfunction primarily due to neuromuscular factors (MPD), in contrast with primary degenerative changes or mechanical derangements of the TMJs.

2. Patients should not be currently receiving another form of TMJ therapy, or previous treatment has stabilized the condition or been ineffectual. An ideal subject would be one who has not been previously treated, but who may be about to begin splint therapy.

3. Patients should not have a history of surgical or other invasive procedures conducted upon the TMJs.

4. The onset of the disorder can be gradual or sudden as in MVA. I have a particular interest in contrasting the treatment outcome of patients involved in personal injury suits, with that of non-personal injury patients.

If you have an appropriate patient for the study, the procedure is as follows:

1. Perform an initial examination and complete my CLINICAL EXAMINATION form.

2. Refer the patient to my office, or ask your receptionist to book an appointment by calling 486-6633.

3. Re-examine the patient at your office approximately 6-7 weeks later following the completion of the study procedures. During this follow-up examination please repeat the clinical examination using a new form to avoid bias.

4. Mail the CLINICAL EXAMINATION forms to my office, or give it to the patient to hand in to us at the debriefing session.

Appendix B

CALL FOR RESEARCH SUBJECTS

BIOFEEDBACK AND RELAXATION TRAINING TREATMENT OUTCOME IN TMJ PATIENTS

Psychologist Roger Gervais is seeking temporomandibular joint pain (TMJ) patients to participate in a treatment outcome study comparing the effect of biofeedback and relaxation training upon this condition. The study is limited to FEMALE TMJ patients only.

Some of the common signs and symptoms of TMJ dysfunction include:

- a sore, tired, or stiff jaw, due to clenching or grinding the teeth;
- crackling, popping, or clicking noises when the jaw moves;
- inability to open the mouth widely;
- pain in the jaw joints;
- frequent headaches or pain in the temples, behind the eyes, or in the neck.

If you experience one or more of these symptoms and wish further information on participating in this research study, please call Roger Gervais at 486-6633. An answering machine will be available to take your call after office hours.

There is no fee for participating in this research project apart from a refundable deposit.

Appendix C

Clinical Examination

NAME: ________________________ DATE: ____________________

OPENING

Maximum opening (between incisal edges) ________ mm
+/- overbite correction ________ mm

Painful to open to maximum 1. YES 2. NO
Protrusive opening habit 1. YES 2. NO

TMJ NOISE (Please complete according to the 0 - 3 scale)

			Scale
	Opening	Closing	0 Negative
Popping	L ___ R ___	L ___ R ___	1 Slight
Soft Tissue Grating	L ___ R ___	L ___ R ___	2 Moderate
Hard Tissue Grating	L ___ R ___	L ___ R ___	3 Severe

PALPATION PAIN (Please complete according to the 0 - 3 scale)

	Without Movement	With Movement
TMJ lateral	L ___ R ___	L ___ R ___
TMJ posterior	L ___ R ___	L ___ R ___

Post temporal	L ___ R ___	Sterno mastoid	L ___ R ___
Ant temporal	L ___ R ___	Suboccipital area	L ___ R ___
Superficial masseter	L ___ R ___	Med pterygoid	L ___ R ___
Deep masseter	L ___ R ___	Lat pterygoid	L ___ R ___
Hyoid, ant gastric	L ___ R ___	Temporal tendon	L ___ R ___

OCCLUSION

R 8 7 6 5 4 3 2 1 1 2 3 4 5 6 7 8 L
Missing Teeth ______________________________________ CD/CD

8 7 6 5 4 3 2 1 1 2 3 4 5 6 7 8

Angle Class Crossbite (describe) ______________
____

Molar Rt. ___ Lt. ___ ____________________________________
Canine Rt. ___ Lt. ___ ____________________________________

Incisal Relationship

Overbite (vertical) _____ mm Overjet (horizontal) _____ mm Anterior contact 1. YES 2. NO

Lateral Guidance
Flat _____ Normal _____ Steep _____

Appendix D

BIOFEEDBACK AND/OR RELAXATION TREATMENT STUDY

As your dentist has explained to you, your jaw or facial pain (TMJ) may be related to a number of factors. Some of these include stress, physical tension, or muscle spasm, which initiate, maintain, or aggravate your condition. TMJ problems are often treated from a multidisciplinary approach.

Your dentist may have taken impressions for a occlusal splint, referred you for physiotherapy, and recommended biofeedback or relaxation training--each of these therapies has an important role to play in the treatment of your condition. If your dentist has referred you for biofeedback or relaxation training, you are invited to participate in a research study coordinated by psychologist Roger Gervais, M.Ed.

The aim of this study is to evaluate the treatment outcome of two types of biofeedback therapy contrasted with relaxation training. Participants will be assigned to one of the three treatment groups or a control group. The total length of the study will be approximately 6 weeks, and limited to female TMJ patients. There will be no cost for participation in the study apart from a $70.00 deposit refundable at the conclusion of the treatment program.

If you wish to be included in this important clinical research project, ask your dentist to complete the enclosed Clinical Exam sheet and call us to schedule an initial appointment. For further information contact Roger Gervais, M.Ed. at 486-6633.

Appendix E

BIOFEEDBACK TRAINING AGREEMENT

I, ____, understand that I have been referred by Dr.____________________________ to Roger O. Gervais, M.Ed., Chartered Psychologist in the Province of Alberta, for biofeedback and/or relaxation therapy as part of my treatment for a temporomandibular joint (TMJ) condition.

I understand that the records and information collected during my treatment will be included in a research project contrasting the outcome of two alternate biofeedback training protocols in the treatment of temporomandibular joint dysfunction (TMJ). All data collected, analyzed, or published as a result of my participation in this biofeedback therapy program will be treated in such a way as to protect my identity. Any communication between Roger O. Gervais, and other disciplines regarding my treatment will respect professional confidentiality and will require my prior written consent.

I understand that the biofeedback therapy will consist of the following program:

1. Initial biofeedback assessment and baseline symptom charting period;
2. Five 1 hour biofeedback training sessions;
3. Home relaxation tape practice, daily, or as specified, for the duration of the biofeedback training program;
4. Symptom charting, four times daily for the duration of the biofeedback therapy program;
5. Completion of MMPI, MBTI, and ACT tests;
6. Follow-up examination with Dr. ________________________.

I understand that the home relaxation practice and symptom charting are essential components of my treatment program, and I assume full responsibility and commitment for completing the specified practice and charting schedules.

I understand that a $70.00 deposit is payable to Roger O. Gervais at the initial biofeedback assessment appointment. The deposit will be refunded in full only upon satisfactory completion of the biofeedback therapy program including:

1. Attendance at all scheduled biofeedback training office appointments;
2. Regular home relaxation practice as specified;
3. Regular and complete symptom charting;
4. Return of all materials including the relaxation tape, symptom chart, and personality tests;
5. Follow-up examination by Dr. ________________________.

I have read, understood, and agree to the terms of this biofeedback therapy agreement.

Signed:_________________________________ Date:___________________________

RELAXATION TRAINING AGREEMENT

I, ______________________________, understand that I have been referred by Dr.______________________________ to Roger O. Gervais, M.Ed., Chartered Psychologist in the Province of Alberta, for biofeedback and/or relaxation therapy as part of my treatment for a temporomandibular joint (TMJ) condition.

I understand that the records and information collected during my treatment will be included in a research project contrasting the outcome of two alternate biofeedback training protocols and relaxation training in the treatment of temporomandibular joint dysfunction (TMJ). All data collected, analyzed, or published as a result of my participation in this therapy program will be treated in such a way as to protect my identity. Any communication between Roger O. Gervais, and other disciplines regarding my treatment will respect professional confidentiality and will require my prior written consent.

I understand that the relaxation therapy will consist of the following program:

1. Initial biofeedback assessment and baseline symptom charting period;
2. Home relaxation tape practice, daily, or as specified, for the duration of the relaxation training program;
3. Symptom charting, four times daily for the duration of the relaxation therapy program;
4. Completion of MMPI, MBTI, and ACT tests;
5. Weekly telephone or office visit progress report;
6. Follow-up biofeedback assessment;
7. Follow-up examination with Dr.______________________________.

I understand that the home relaxation practice and symptom charting are essential components of my treatment program, and I assume full responsibility and commitment for completing the specified practice and charting schedules.

I understand that at $70.00 deposit is payable to Roger O. Gervais at the initial biofeedback assessment appointment. The deposit will be refunded in full only upon satisfactory completion of the relaxation therapy program including:

1. Regular home relaxation practice as specified;
2. Regular and complete symptom charting;
3. Weekly telephone or office visit progress report, and attendance at all scheduled appointments;
4. Follow-up biofeedback assessment;
5. Return of all materials including the relaxation tape, symptom chart and personality tests;
6. Follow-up examination by Dr.______________________________.

I have read, understood, and agree to the terms of this relaxation therapy agreement.

Signed:______________________________ Date:____________________

CONTROL GROUP AGREEMENT

I, ______________________________, understand that I have been referred by Dr.______________________ to Roger O. Gervais, M.Ed., Chartered Psychologist in the Province of Alberta, for biofeedback and/or relaxation therapy as part of my treatment for a temporomandibular joint (TMJ) condition.

I understand that the records and information collected during my treatment will be included in a research project contrasting the outcome of two alternate biofeedback training protocols and relaxation training in the treatment of temporomandibular joint dysfunction (TMJ). All data collected, analyzed, or published as a result of my participation in this therapy program will be treated in such a way as to protect my identity. Any communication between Roger O. Gervais, and other disciplines regarding my treatment will respect professional confidentiality and will require my prior written consent.

I understand that I have been assigned to the CONTROL GROUP condition which will consist of the following program:

1. Initial biofeedback assessment;
2. Symptom charting, four times daily for the five-week duration of the control program;
3. Weekly telephone or office visit progress report;
4. Follow-up biofeedback assessment;
5. Follow-up examination with Dr.______________________.

I understand that the symptom charting is an essential component of my participation in the treatment study, and I assume full responsibility and commitment for completing the specified charting procedures.

I understand that a $70.00 deposit is payable to Roger O. Gervais at the initial biofeedback assessment appointment. The deposit will be refunded in full only upon satisfactory completion of the study program including:

1. Initial biofeedback assessment;
2. Regular and complete symptom charting;
3. Completion of MMPI, MBTI, and ACT tests;
4. Weekly telephone or office visit progress report;
5. Follow-up biofeedback assessment;
6. Return of all materials including symptom chart and personality tests;
7. Follow-up examination by Dr.______________________.

I understand that upon completion of my control group involvement, I have the option of receiving five biofeedback and/or relaxation training sessions at no extra cost.

I have read, understood, and agree to the terms of this biofeedback training study.

Signed:______________________________ Date:________________

Appendix F

TMJ Screening Questionnaire

1. Do you clench or grind your teeth during the day?
2. Have you been made aware of clenching or grinding your teeth during the night?
3. Do you have chronic headaches, or neck and shoulder pains?
4. Do you frequently have gastro-intestinal disturbances?
5. Do you ever wake up with an awareness of, or about, your teeth or jaw like you had them clenched in your sleep?
6. Do you have any awareness of the muscles of your neck or shoulders?
7. Do you have a tight or stiff neck?
8. Do you now, or have you ever had, pain in your jaw joints or the sides of your face (in and about the ears)?
9. Do you have a clicking jaw joint or have you ever experienced an inability to move your jaw or open your mouth widely?
10. Which side of your mouth do you chew on?
11. Do you tend to breathe mostly through your nose?
12. Are you aware of persistend ringing in your ears?
13. Have you ever experienced pain or burning sensations in your:
    a. neck; b. shoulders; c. back; d. hips
14. Have you ever had treatment for problems of your:
    a. neck; b. shoulders; c. back; d. hips
15. Have you ever been told you have:
    a. scoliosis; b. lordosis (swayback)
16. Have you ever been told you have a leg shortening on one side?
17. Have you ever suspected a leg shortening on one side?
18. Is your condition the result of an injury or accident?
19. Are you presently involved in legal action regarding your condition?
20. How long have you had jaw problems?

Appendix G

Symptom Chart Name: Start Date:

	1				2				3				4				5				6				7			
5																												
4																												
3																												
2																												
1																												
0																												
	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e

	1				2				3				4				5				6				7			
5																												
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1																												
0																												
	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e

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	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e

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0																												
	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e	b	l	s	e

The following six point scale is useful in helping people monitor the severity of their symptoms:
0: No pain.
1: Low level, only enters awareness when you think about it.
2: Aware of pain most of the time, but it can be ignored at times.
3: Painful, but still able to continue normal activities.
4: Severe pain, difficult to concentrate on normal activities.
5: Intense incapacitating pain, unable to continue normal activities.
To monitor your symptom level mark the appropriate number on the graph at each of the four times indicated and join the points together. B: Breakfast L: Lunch S: Supper E: Evening

Appendix H

EMG Assessment Procedure

The pre and posttreatment EMG assessment component of the present study consisted of a number of stressors during which repeated measures of EMG activity were obtained. The procedure was conducted as follows:

1. 15 minute introduction and hookup
2. 6 minute resting baseline on four channels:
   Ch.1: left temporalis
   Ch.2: left masseter
   Ch.3: right temporalis
   Ch.4: right masseter
3. 2 minute serial subtraction
4. 1 minute physical exertion (legs extended)
5. Cracker mastication, followed by 3 minute relaxation
6. 10 minute cleanup

At the outset of the EMG assessment, the patients were given a brief introduction to the nature and purpose of the electromyographic recording after which the EMG electrodes were attached. The sequence of the assessment and the particular stressors were not disclosed ahead of time. Subjects were led into the six minute baseline with the following verbal directions:

> We will be starting the experiment shortly and I would ask you to sit quietly with your arms in a comfortable position, and both feet flat on the floor (a foot rest was provided if necessary). I would like you to maintain this position with your eyes closed for the duration of the recording

> periods, and please refrain from any extraneous movement. Unless you have any questions, I will begin the first recording period now. It will last for six minutes.

After completion of the baseline period, the serial subtraction stressor was introduced:

> Stop! Now, I want you to take the number 1000, subtract 7 and get an answer. Subtract 7 again, get a new answer, and so on. Do this as fast as you can, silently, and without making any mistakes. I will ask you for your answer when the time is up. Do you understand the task? You may start now.

After the serial subtraction exercise:

> Stop! The time is up. How far did you get? Now I would like you to forget about the subtraction and extend your legs straight out in front of you and squeeze them together as hard as you can. Continue doing this until I tell you to stop. This will last for one minute.

At the end of the sixty second recording period: "Stop! Now just return to relaxing with your feet flat on the floor."

The patients were then given a small cracker with these instructions: "I would like you to eat this cracker and wash it down with a drink of water. When you have finished, just return to relaxing." When this last time period had expired, subjects were told the initial recording procedure was over and the electrodes were detached.

Appendix I

Relaxation Script

*Asterisks indicate where to pause for periods upwards of 5 seconds.

Make yourself as comfortable as you can. Close your eyes. Now stretch your legs as far as they can go and turn the toes under and tighten the muscles very tight and hold it.* Now, also tighten the muscles in your calves and those in your thighs. Make your entire leg as tight as a drum and hold it.* Hold it.* And now relax all the muscles in your toes, all the muscles in your calves, all the muscles in your thighs. Let your legs go completely limp. And now feel that wonderful relaxation coming up from your toes, up your calves, up your thighs and you are feeling wonderfully relaxed, beautifully relaxed, very calm, very relaxed.* Now I want you to stretch out your hands and make a fist. Feel the tightness. And now make it tighter, tighter, tighter, and hold it.* And now also tighten the muscles in your wrists, in your forearms, in your upper arms.* Hold it,hold it.* Now let go and get the wonderful feeling of relaxation right through your fingers, your hands and now through your forearms and your upper arms. Let your arms go completely limp, and you are feeling wonderfully relaxed, beautifully relaxed, very calm, very relaxed and feeling just beautiful.* Now I want you to arch your back backwards, raise your chest, tighten your neck and shoulder muscles and your stomach muscles but keep breathing regularly. Make all those muscles as tight as you can, tighter, tighter, and hold it, hold it.* All right, now let go, just let go and you get that wonderful feeling of relaxation. Just feel the muscles relax from your back, from your shoulders, from your chest, from your stomach; all over your back. And all the muscles are feeling wonderfully relaxed.* Now I want you to tighten the muscles in your face. Make a funny face. Tighten the muscles around your mouth, the muscles in your chin, around your eyes and your forehead. Wrinkle your brow. Make them tighter, tighter, tighter, hold it, hold it.* All right , now let go just let go. Let go and get that wonderful feeling of relaxation from all the muscles in you forehead, the muscles around your eyes, the muscles of your cheeks, the muscles of your chin, and the muscles around your mouth. And you are feeling wonderfully relaxed, beautifully relaxed. Very calm and very relaxed, wonderfully relaxed.* Now take a very deep breath and hold it. Hold it, hold it.* Now slowly let it out and you are letting out all your tensions, your frustrations, your anxieties and feeling wonderfully well.* Once again, take a very deep breath and hold it, hold it, hold it.* Now slowly, slowly let it out and relax your tensions, your frustrations, your anxieties and you are feeling wonderfully well, wonderfully well.* We will now proceed to relax every part of your body progressively. And while we are doing this, you will hear my voice clearly and distinctly. You will be aware of your surroundings, although you may care less and less about what goes on around you.* Now, direct your thoughts to the top of your head, your scalp and think that whatever tension exists there is rapidly vanishing. Your scalp is becoming less and less tight and the top of your head is becoming completely relaxed.* And now think of your forehead and let all the muscles in your forehead relax and become loose and limp.* And now your eyes, and all the small muscle groups around your eyes. Just allow then to become loose and limp and relaxed. And just relax more and more and let yourself go completely.* Now your facial muscles, the muscles in your cheeks and around your nose and the muscles around your mouth and teeth. Just let them all relax and let them all go very loose and limp.* Now relax the muscles in your throat area, your speech mechanism and your swallowing apparatus and all the other muscles in your neck and just allow them all to relax.* Now think of your shoulders, and permit your shoulder muscles to relax.* Relax the muscles in your upper arms, your elbows and your forearms and all the muscles up and down your arms. Just let them go loose and limp.* Your wrists, your hands, your very fingers; completely relaxed.* And now your chest. Relax all the organs and muscles within your chest.* And the muscles in your stomach area, your abdomen and all the muscles and organs within that region. Allow them to become flaccid and relaxed.* And the pelvic region fully relaxed.* Now think of your back and all the muscles up and down your back, the long muscles and the small muscles up and down your spine and let all these muscles relax completely.* Now think of your thighs and relax all the muscles there, your knees and the calves of your legs. And just let all those muscles up and down your legs relax completely.* Your ankles, your feet, your toes.* Relax now and let yourself go completely. Just go limp all over. Permit every organ and every fibre of your body to become completely and profoundly relaxed. It feels so restful, so pleasant to be completely relaxed. You hear my voice clearly and distinctly. But nothing else seems to matter to you.*

Your arms and legs, if you will think of them for a moment, feel rather heavy and they are so relaxed and also quite numb and dull, though quite pleasantly so. In fact, your entire body feels heavy in this position. Heavy, so heavy, that it seems it would require a superhuman effort to move a muscle.* Now I am going to count down from ten to one and while I am doing so, I want you to think of a scene that makes you feel calm, that makes you feel relaxed and that gives you a feeling of well-being. Think of that scene in all its details. Put yourself into it and let yourself relax more and more deeply and profoundly and enjoy the calm and peace of that wonderful scene.* Now, with your eyes closed, see that scene in all its details and as I'm counting down from ten to one you are going to find yourself deeper and deeper relaxed and you will have a feeling of well-being.* Calm and relaxed and wonderfully well. Very relaxed.* Ten - just let yourself go completely now.* Nine - deeper and deeper relaxed.* Eight.* Seven - very deeply relaxed.* Six - deeper and deeper.* Five - deeply relaxed.* Four - very calm and very relaxed.* Three - deeper and deeper.* Two - deeper.* And one - very deep and sound relaxation. Think of nothing but relaxation. Feeling wonderfully relaxed. Calm, feeling wonderfully well. And now, like a wave, feel that relaxation spreading from your toes, up your calves, up your thighs, and into your stomach and chest muscles. And now its spreading from your fingers, up your arms, into your shoulders, neck and head region, relaxing every part of your body until you are involved in a wonderful feeling of relaxation.* You are feeling calm, feeling relaxed, feeling like you are floating on a sea of tranquillity; completely calm and at ease.* And now, just enjoy that wonderful feeling of relaxation and well-being for a few moments.*** Soon, I will count to five and you will open your eyes. You will continue feeling as calm and wonderfully in control of your feelings as you feel now. Your mind will be clear and alert and your body will feel very well in every way. You will look forward to your next relaxation session. One - feeling fine. Two - coming up now, feeling very good. Three - feeling very relaxed, but alert. Four and five - open your eyes, feel relaxed, feel calm, feel wonderfully well (Feil, 1982).

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Appendix J

Biofeedback Training Procedure

The biofeedback training sessions followed the same format for the weekly and daily biofeedback patients. At the outset of the session, each patient's progress and symptom chart was reviewed and any questions regarding the charting or home practice were answered:

> Before we get you hooked up, tell me how your week/day went. How did your relaxation practice go? How often did you use the tape? Did you have any problems with the charting, and did you complete it regularly? Do you have any further questions?

The application of the electrodes proceeded as documented above. A six-minute resting baseline recording was obtained before the four training trials. As the Biocomp telemeter was turned on the following instructions were provided:

> As the system warms up, I want you to sit quietly in the chair with your eyes closed. During this first run we will simply be monitoring your muscle tension to see where you are starting from. Are you ready? I will begin recording now and leave the room for six minutes.

At the conclusion of the baseline recording the graphs and values for each muscle site were displayed, explained, and the biofeedback procedure was introduced:

> These are the readings for each of your temple and jaw muscles. As you can see, the left/right temple/jaw muscle(s) is/are more tense than the others. I will set the display so that the highest muscle is graphed on the screen. The tension levels in the other muscles are displayed digitally at the bottom of the screen. If you want, you can also follow the tone which varies proportionally

> with the amount of tension in the muscle. Try to bring the tone as low as possible. During your training I want you to try and reduce the height of the graph as much as possible and try to balance the four numbers at the bottom of the screen so that the muscle tension is even in your face and jaw. For the first few minutes feel free to experiment and see what happens when you move your jaw around. Once you have a sense of controlling the muscles, focus on reducing and balancing the tension in your muscles by bringing the graph and numbers as low as possible. Do you have any questions? This training run will last for six minutes. I will be in and out of the room to check on how you are doing. Are you ready to begin?

After the initial training trial, the channel means were again displayed and the patient was encouraged to "try to see if you can bring the tension down even more." During the last training trial of the session the patient was instructed: "Try to memorize just how your jaw and temple muscles feel during this last run. This is important so that when you get home and do your relaxation practice, you can re-create the feeling of relaxation you had during the biofeedback session. This will help you to apply your biofeedback skills in your everyday life."

At the conclusion of the biofeedback training session the electrodes were removed, the next appointment was confirmed, and patients were encouraged to be consistent with their symptom charting and home relaxation practice.

Appendix K

No.

POST-TREATMENT QUESTIONNAIRE Name:

Date:

1. My TMJ symptoms following the biofeedback and/or relaxation training program are:
    1. Unchanged
    2. Improved
    3. Worse

2. My TMJ symptoms have improved by _____ percent.

or

3. My TMJ symptoms have worsened by _____ percent.

4. I used the relaxation tape _____ times per week.

5. Which of the following did you find the most useful throughout the biofeedback/relaxation training program:
    1. Biofeedback training
    2. Home relaxation practice
    3. Symptom charting
    4. Other (describe):

6. Overall, has your participation in this program made a significant difference in your condition?
    1. Yes
    2. No

7. Do you plan to continue using your relaxation skills on a regular basis?
    1. Yes
    2. No

Appendix L

Referral Acknowledgement Letter

Dear Dr.:

Thank you for referring ______________ as a participant in my TMJ treatment outcome study. She has been assigned to one of 3 treatment conditions or a no-treatment control condition. Her involvement in this study will be completed in approximately 6 weeks, __________, 1989, at which time I will instruct her to present again to your office for the post-treatment evaluation. If you wish, you may then give both Clinical Examination forms to her so she can bring them to the debriefing session.

I will be contacting you with a brief summary of the treatment outcome at the conclusion of ______________'s participation in the study.

Sincerely,

Roger Gervais, M.Ed.,
Chartered Psychologist.

RG/lr

Appendix M

Ankylosis: a. bony b. fibrous. Bony ankylosis the union of the bones of a joint by proliferation of bone cells, resulting in complete immobility. b. Fibrous ankylosis reduced mobility of a joint due to proliferation of fibrous tissue (Friel, 1974, p. 99). These are the most common forms of ankylosis found in the temporomandibular joint.

Articular Disc: a pad of dense fibrous tissue that is nonvascular and noninnervated except in the peripheral areas, lying between the condyle of the mandible and the cavities and provides an articulating surface between the condyle and fossa. Also referred to as the **Meniscus** (Bell, 1982; Ermshar, 1982).

Bruxism: rhythmic or spasmodic grinding of the teeth in other than chewing movements of the mandible, especially such movements performed during sleep. Dental malocclusion and tension-release factors are the usual inciting causes (Friel, p. 231).

Condyle: the rounded projection on a bone, usually for articulation with another. Here, the articular surface of the mandible upon which rests the articular disc (Friel, p. 350; Hylander, 1980).

Crepitus: scraping or grading sounds produced in the joint upon articulation. Often related to perforation of the articular disc resulting in bony contact.

Meniscus see Articular Disc

Muscle Spasm: a sudden involuntary contraction of a muscle or group of muscles that are functionally related. It is attended by pain and interference with function, and it is manifested by involuntary rigidity, distortion, or movement. Clonic muscle spasm is of momentary duration; tonic spasm persists for a period of time. Cycling muscle spasm is protracted tonic spastic activity that become self-perpetuating, presumably as the result of pain incidental to continued spastic contraction of the muscle. Isometric spasm causes muscular rigidity with marked resistance to stretch; isotonic spasm causes shortening of the muscle, which produced distortion or skeletal movement (Bell, p. 57).

Muscle Splinting: a protective mechanism whereby a threatened or injured component of the musculoskeletal system is immobilized by increased tonicity of its surrounding musculature. The condition usually subsides when the threat or injury disappears. Prolonged splinting may lead to muscle spasm (Bell, p. 57).

Muscle Tonus: the resistance of the muscle to elongation or stretch. Hypertonicity refers to a relative increase in passive resistance to stretching the muscle; hypotonicity refers to a decreased passive resistance to stretch. Muscle tonus serves two purposes: (1) it furnishes the muscular activity needed to maintain sharp contact of the articulating parts in joints when at rest or under negative interarticular pressure imposed by the effect of gravity, and (2) it maintains the muscles in an optimum state of readiness for contraction (Bell, pp. 56-57).

Tinnitus: a noise in the ears, as ringing, buzzing, roaring, clicking, etc. Such sounds may at times be heard by others than the patient (Friel, p. 1613).

TMJ: the temporomandibular or jaw joint. Often used alone as an abbreviation for the temporomandibular joint pain-dysfunction syndrome.